ARCHIVES OF OPHTHALMOLOGY.

A USEFUL ADDITION TO THE TEST CHART.

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WHERE a fundus is apparently normal, where in addition the vision is normal, the oculist in his routine office or clinic work usually does not make a perimetric examination of a patient. He does, however, I am sure, often therefore overlook certain anomalies, and none I think more frequently than small central scotomata for red or green.

For a number of years, I have been trying to evolve some rapid, practical method of examination in connection with the test-chart investigation, by means of which, as a routine practice, we may at least roughly test each patient's central color sense, when we try his visual acuity, and often in this manner pick out certain patients for further and more careful perimetric examination and find anomalies that we might otherwise have overlooked.

The end toward which I have striven has been to add to the ordinary test chart small red, green, and blue squares of such size that every normal eye should be able to differentiate them correctly at the ordinary test distance of six metres, and still they should be so small that where color defects, even though not marked, were present, I should be able to discover this fact by means of this test. Of course, I did not intend this as a test which should in any way replace perimetric examination, but merely as one which might help me select such cases where a perimetric examination was necessary.

Great as is the variation in the visual acuity of different individuals having perfectly normal eyes, so there is fully as large if not larger variation in the visual angle for colors.

It is not necessary here, nor of any especial benefit, to enter into a detailed account of the literature on the subject. I shall, however, briefly mention the studies of Aubert and also those of V. Wittich, whose work in determining the visual angle for colors I used as my own basis of comparison.

Aubert [1] testing himself with colored squares 10*mm* in size found it very difficult to determine the visual angle for different colors, because when the visual angle becomes very small, different colors assume different tones and nuances. I omit his findings on all colors except such as interest us in connection with this subject.

Red on black ground at 59″ appeared red, on white ground at 59″ black, at 1′, 43″ it showed color.

It might be well, for me, to interpolate here that though it is commonly accepted that the smallest visual angle at which objects can be differentiated is 1′, this is not true for colors, as is shown by the fact that the fixed stars, whose visual angle is too small to measure, show color.

Aubert further found that ultramarine blue on black ground at an angle of 1′, 14″ appeared gray, at 4′, 17″ blue, on white ground at 1′, 8″ black, at 5′, 43″ blue. Bright blue and bright green at 1′, 8″ appear gray, at 2′ they can be differentiated as blue and green; at 1′, 49″ bright blue on white ground is deep blue.

V. Wittich [2] using squares 2*mm* in size on white ground in bright daylight found the following angles for himself: red, 2′, 50″; bright green, 1′, 43″; dark green, 13′, 46″; light blue, 2′, 17″; dark blue, 2′, 17″. My own tests were made in the following manner: three squares 2*mm* in size, red, bright green, and deep blue, were pasted on to the white background of a test chart illuminated after the usual fashion with reflected electric light, and at such a height as to be as nearly as possible on a level with the eyes of the individual to be tested, the patient, with one eye covered, not knowing the color of the squares, stood at a distance of eight metres.

[1] *Abhandl. der Schlesich. Gesellschaft, Bres.*, p. 74.

[2] *Med. Jahrbuch*, Bd. 72, p. 37.

He now approached the test chart step by step until he was able to correctly distinguish each square.

Testing one hundred different individuals with normal eyes after this fashion, I found an astonishing difference in the visual angle for colors of different persons; I even found differences in the visual angle of the same individual when tested at different times.

Women, as a rule, had a smaller visual angle for colors than men; bright children from twelve to eighteen years of age could usually distinguish the squares sooner than older people. The visual angle differed with the length of time of observation. Green at first glance seemed to reveal its color more quickly than red; the patient at the first glance often called green correctly, after prolonged gaze he was often in doubt; red, on the contrary, showed its color more clearly after prolonged observation.

Strange to say, in perfectly normal eyes color acuity is by no means directly dependent on visual acuity. I often found that individuals with remarkably keen sight could not distinguish colors as well as others whose vision was less keen.

Averaging my results in these one hundred different individuals, I found the average distance at which the red square was correctly named to be 4½ metres. Many individuals correctly named the color at a distance of almost six metres, others had to approach to within less than three metres before they could with certainty name the color as red.

In the same manner I found the average distance for green to be a little less than four metres. Strangely enough a number of individuals correctly named the green square before they could the red; the vast majority however correctly named the red before they could distinguish the green.

The distance at which the green square could be named correctly varied from almost six metres to two and one half metres.

My results, therefore, for red and green coincide with those of Aubert rather than those of V. Wittich, Aubert finding the angle for green (2′) to be greater than that for red (1′, 43″), while V. Wittich found the angle for red (2′, 50″)

greater than green (1′, 43″). My illumination, however, being different, easily accounts for a difference in findings.

The average distance at which these patients were able to say with certainty that deep blue was not black was at 2¼ metres.

The following are the results which I have obtained after repeated tests on myself, though knowing what the colors were may have somewhat influenced the results.

At eight metres the squares are seen as small spots of varying shades of blackness.

At seven metres blue is black, green is gray, red is assuming some color, but it is impossible to say what that color is.

At six metres blue is black, green is decidedly gray; red shows more color but it is still impossible, with certainty, to say what that color is—it might be brown, orange, or red.

At five metres I am able to distinguish red with certainty, green might be either light blue or green, blue is black.

At four metres I can be sure of red and green, blue is black.

I must approach to within a little less than three metres before I can be sure of blue. While I was trying to determine the color angle for normal eyes on the test chart, under customary methods of examination, I had also in the meantime been trying these same tests on a number of individuals for whose benefit this test was being devised, *i. e.*, individuals with small central color scotomata for red and green, and I soon found that though it was necessary and useful to determine an average standard for normal eyes, it was by no means necessary to be absolutely guided by this standard in differentiating those cases where scotomata were present, because their visual angle for red or green was so markedly altered as to be easily detected even though the squares used were considerably larger than those necessary in determining the average color range of normal eyes; *e. g.*, I found that in no instance where a central scotoma for red was present was a patient able to distinguish on the test chart at a distance of 6 metres a red square 5*mm* in size; the same was true where a scotoma for green was present, no patient being able in such a case to distinguish a 5*mm* square of that color at a distance of 6 metres. I have therefore for myself adopted the following plan.

I have affixed to my test chart four squares as follows: one red square 2½*mm* in size and one red square 5*mm* in size, one green square 3*mm* in size and another green square 6*mm* in size, the test being made at 6 metres.

When a patient cannot see the smaller squares which mark the average color angle according to my tests, I am suspicious of a color defect; when he cannot differentiate the larger ones I am certain that it is a case for careful perimetric examination. It is also a good plan to have four squares rather than two, inasmuch as it eliminates guesswork much more certainly.

Where these colored squares are affixed to the test chart, each patient's color angle can be determined by the examining oculist with no appreciable loss of time.

The addition of the blue square to my test chart though of experimental interest, I have found of no practical value.

Before concluding this article I should like to give a very brief history of four cases in each one of which I detected a color scotoma by means of this test.

CASE 1.—Mr. I. G. W., aged 38, referred to me by Dr. L. Greensfelder because of headaches and asthenopia. Patient had been recently operated upon for appendicitis. With proper glass patient had a vision of $\frac{6}{6}+$. Fundus was apparently normal. Media clear. Patient has been a steady but not excessive smoker for many years. He averages about six cigars daily. Most moderate in the use of alcohol.

At a distance of 6 metres he is unable to tell the color of either the green 6*mm* or red 5*mm* square.

Careful perimetric examination revealed a small central scotoma both for red and green.

Smoking was stopped and patient given strychnia treatment by Dr. Greensfelder. In six weeks the scotoma had absolutely disappeared and patient was able, without the least difficulty, to correctly name both the smaller red (2½*mm*) and green (3*mm*) squares.

I must frankly confess that had it not been for the red and green squares of the test chart, I should in this case in all probability have overlooked the scotoma, as there was nothing else about the case to arouse my suspicions.

CASE 2.—L. R., 47 years of age; distiller; consulted me

because of presbyopia. With a +.50 sph. in each eye patient had normal vision. Fundus apparently normal, media clear. Large green square on test chart was named correctly. Red was called brown. Investigation disclosed the fact that the patient was a moderate user of alcohol, but a heavy smoker. Perimetric examination disclosed well-marked oval central scotoma for red; there was no other symptom present.

CASE 3.—H. H. G., shoe salesman, 40 years of age. Patient consulted me because of difficulty in reading on trains. Patient confessed to syphilitic infection thirteen years ago. With proper glasses vision is normal in right eye, slightly below normal in left. Patient was unable to tell color of either red or green 5*mm* square on test chart.

Patient confesses to being a steady drinker and a constant chewer of tobacco.

Thorough physical examination by Dr. M. L. Goodkind disclosed no further abnormality. Wassermann test was negative; on removal of tobacco and exhibition of strychnia scotoma gradually disappeared.

CASE 4.—Was a diabetic woman 59 years old who came to me merely to have her glasses changed. For the past five years she has been on a careful diet; ophthalmoscopic examination revealed a possible slight pallor of the disk. Patient's vision in each eye with proper glass was normal.

Patient can distinguish both the large and small green square at 6 metres; entirely unable to name color of large red square at same distance. Perimetric examination showed well-marked central scotoma for red.

CASE 5.—After finishing this article, the following case came under my observation. Mr. G. M., 47 years of age. Referred to me by Dr. L. E. Frankenthal. Patient had syphilis twenty-three years ago; for the past three years has suffered from diabetes; for many years has been an excessive smoker, is moderate in the use of alcohol. Several months ago had several attacks of epilepsy of Jacksonian type, convulsions beginning in left hand; since that time there has been no recurrence of convulsions, energetic mercury treatment having been used. Wassermann test at present is positive.

Vision in each eye is normal. Ophthalmoscope disclosed nothing abnormal. Pupils are slightly unequal, both react to light and accommodation though somewhat sluggishly.

Patient was not able to correctly name either the large red or green square on the test chart.

Perimetric examination disclosed normal color field for

white, slightly contracted fields for blue, red, and green, well marked central scotoma for red and green.

Conclusion.—We have in small red and green squares, of a size such as I have indicated, affixed to the ordinary test chart a valuable and time-saving aid in the detection of central color scotomata for red and green.

TWO CASES OF INTERSTITIAL KERATITIS OF SYMPATHETIC OR SYPHILITIC ASPECT.

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WHILE attending the following case I read in the Report of the French Ophthalmological Society, May, 1910, about a similar case described by Dr. Armaignac of Bordeaux, France, under the following heading: "A Case of Interstitial Keratitis of Sympathetic Appearance Due to a Slight Traumatism of the Eye. Medico-legal Deductions." A girl of 21 with no specific history suffered a slight burn of the cornea, with some drops of melted lead. Three days after the accident, which caused very little local reaction, an interstitial keratitis developed, and eight days later vision was completely lost as a consequence of the corneal opacity. This opacity kept on increasing during eight days more, and then the periphery of the cornea began to clear, but the right eye, until then well, became also affected, and very soon it was in the same condition as the left one. The left, the originally involved eye, then began to get well and the cornea cleared very rapidly; there remained only a slight corneal opacity due to the burn. The right eye also rapidly improved, but there remained in the middle of the cornea an opacity that did not disappear in spite of all efforts. Vision in both eyes was reduced to $\frac{6}{10}$. During the course of this keratitis the patient suffered from an attack of acute rheumatism lasting six weeks. Dr. Armaignac gave the patient a certificate stating that she was cured without paying attention to the condition of the eye that became last affected. The patient, however, insisted that the insurance company should indem-

nify her on account of the defect of the eye last affected, because she thought that it was due to the burn suffered by the left eye. An expert was called and his opinion was the same given by Armaignac. The patient has not stated whether she is ready to accept the decision of the expert, and Armaignac asks if in case of appeal to the law courts, the condition of the eye last affected should be regarded as a sympathetic ophthalmia, or should the rheumatism be regarded as the cause of the keratitis in both eyes.

The question of possible sympathetic ophthalmia in the case published by Armaignac is also suggested by the one which we are going to describe, because there was an old scar in the ciliary region of one eye following a corneo-scleral wound that had occurred some years before. In our case a doubtful specific history suggested the treatment; cases of interstitial keratitis with a specific history provoked by traumatism are not infrequent according to Antonelli.

S. B. M., a boy, 3 years old, was brought to us in 1888, and we were told that the baby, while playing with a bottle of seltzer, had fallen to the floor, the bottle exploded, and a piece of glass injured his right eye. The wound involved the cornea, iris, and ciliary body within, extending from the upper and outer margin of the cornea without touching the border downwards and inwards through the ciliary region. The wound healed and the eye was quiet when the patient was discharged.

On August 3, 1907, the patient returned, stating that after the accident he had been two years without any trouble, but then the eye became inflamed and painful. There was injection of the bulbar conjunctiva, palpebral œdema, and intense pain. We feared a panophthalmitis and began to think of enucleation, which had already been proposed by others to the patient, fearing sympathetic ophthalmia. We ordered ice to be applied to the eye and other therapeutic agents; the most alarming symptoms disappeared and the patient returned home.

In August, 1909, he injured his good eye, the left, with a piece of wood. On examination there was injection of the bulbar conjunctiva, contracted pupil, cloudy aqueous humor, and intense periorbital pain. I diagnosed iritis. The pupil was dilated with atropin in a transverse oval form. I began again to fear the presence of sympathetic ophthalmia but the inflammatory symptoms ceased after one month and the patient left.

September 20, 1910.—The patient came again for consultation with iritic symptoms in the left eye. My previous fears of sympathetic ophthalmia recurred again, and I thought seriously of enucleating the right eye, which was the first injured, but finally I limited myself to treating the left eye for iritis. The next day the patient was brought to me completely blind. He thought the loss of sight was due to the atropin. The pupil was half dilated; the aqueous humor very cloudy. Tension not increased. A peripheric interstitial keratitis began to appear. I did not believe myself justified in performing an enucleation of the eye first wounded because it still had perception of light and because there was no pain and it was not inflamed. We ordered mercurial inunctions.

From September 20th to 23d, the symptoms did not change. The keratitis invaded the whole of the cornea, which had a whitish-blue appearance. The treatment (ice, atropin, and mercurial inunctions) was kept up and the family was informed of the gravity of the case and of the possible existence of a sympathetic ophthalmia which would require the enucleation of the right eye previously injured.

September 25, 1910.—The upper part of the left cornea begins to clear.

September 30, 1910.—The keratitis is disappearing and the danger has ceased.

Some days later the cornea completely regained its transparency, and the pupil is almost completely dilated by atropin.

Treatment was continued, except the ice, for three or four weeks.

November 22, 1910.—The patient has had perfect vision in the left eye for one month.

Remarks.—I believe that there was no sympathetic ophthalmitis but simply an interstitial keratitis of the left eye in a patient who had lost the other eye from a wound in the ciliary region.

The keratitis of the left eye was due of course to the traumatism it suffered and which left it predisposed to inflammation. Although the specific history of this patient is doubtful, he may be regarded as a parasyphilitic and as such we may regard the keratitis, considering that many do not believe it to be of specific origin. As a precaution I prudently insisted on mercurial inunctions and would have used subcutaneous injections of mercury if they had been necessary.

OPTIC ATROPHY FROM TRAUMATIC ASPHYXIA, WITH REPORT OF A CASE.[1]

By FRANK JUDSON PARKER, M.D.

TRAUMATIC asphyxia is a forcible compression of the thorax, with respiration suspended for a period sometimes of several minutes, resulting in a discoloration of the head and neck. The number of these cases that have recovered and been studied in life is small. It has long been noted that there was a peculiar discoloration of the face and neck in people who had been crushed to death in crowds and mobs. Ollivier (1) examined the bodies of twenty-three persons crushed to death in the Champ de Mars crowd, and found the violet tint of the face and neck in all of them. Autopsies were done on nine of these in which there was extravasation of blood under the conjunctiva, and Villemin states that there were no indications of cerebral hemorrhage in any of them. Aside from the appearance of the face and eyes, no pathological lesion had been found. Previous to the report of Beach and Cobb (2) our entire knowledge of this condition was from post-mortem examination, and the cause was very obscure. At the time of their report, 1904, only six cases had been recorded in the literature; one by Burrell and Crandon (3) of Boston, and five cases by German observers. Henning Rönne (4) in a very complete review of the subject, published in 1910, found, in all the literature, only sixty recorded cases of traumatic asphyxia. In 12 cases (including his own) disturbances of vision set in without ophthalmoscopic changes. The history of my patient is as follows:

[1] Read before the Section on Ophthalmology, N. Y. Academy of Medicine, December, 1910.

Benjamin Jones, aged 23, was admitted to the Presbyterian Hospital, Dr. Elliot's service, on Nov. 20, 1909, with the following history: While fixing an elevator door, the weights of the elevator, weighing 16,000 pounds, fell, striking his left shoulder. The elevator was stopped automatically, so he was subject to the pressure only for a fraction of a second. He was brought to the hospital, his face black with a sharp line of demarkation running around the neck just over the clavicle. This line separated the darkened area above from the normal below. He was conscious, had marked dyspnœa, total loss of vision in right eye, normal vision in left eye, exophthalmos both eyes, more marked in right, great swelling of the conjunctiva from extravasation of blood beneath. Ophthalmoscopic examination showed a white nerve, right eye, with great engorgement of the veins without tortuosity, slight retinal œdema, no perception of light, sluggish pupillary reaction, tension normal. The left eye, vision normal, retinal œdema, engorgement of the veins, nerve normal. I examined the patient frequently until he left the hospital on Dec. 5th, four weeks after the accident, and at that time the right eye had no perception of light, no pupillary reaction, retina normal, veins enlarged, but much less in size than when first examined, optic nerve atrophic, with central cupping. The left eye was normal except for venous engorgement.

He had no control of the sphincter for a few hours after admission. Temperature ranged from 98.6° to 100.5° F. Respiration 18 to 20. Pulse from 64 to 90. The discoloration disappeared rapidly after the fourth day, and on his discharge from the hospital there remained only a few reddish-black spots on the face and neck, and the subconjunctival ecchymosis.

The cause of the discoloration has been the subject of much interest and discussion, and numerous theories are advanced. Herter experimented on rabbits, and advanced the mechanical theory that venous hemorrhage was found only in the face and neck, being due to the lack of valves in the facial veins, and is more common in young people, who have more numerous skin capillaries, than the old. Burrell and Crandon held that the discoloration was due to a stasis of carbonized blood in the temporarily paralyzed and dilated capillaries. Beach and Cobb were the first to establish the true cause of the discoloration, by studying sections of tissue from the living subject; their conclusions were that it was due to

stasis from mechanical over-distension of the veins and capillaries, with or without paralysis from engorgement of, or pressure on, sympathetic nerves, and the sharp line of demarkation was, according to Perthes's (5) theory, due to the lack of valves in the jugular and facial veins.

Summary of cases:

I will briefly mention the eye conditions noted in the fellowing cases reported.

Braun's (6) case had marked extravasations of blood under both conjunctivæ, pupils dilated and did not react to light.

Herter and Vogt noted only the subconjunctival hemorrhages.

Perthes's (5) case had exophthalmos, conjunctiva completely suffused with blood; patient stated that vision was completely lost for a brief time, returned normal. Perthes considered that this was due to venous hyperæmia, with retinal œdema, and compression of the optic nerve by retrobulbar effusion of blood.

Burrell and Crandon's case. The pupils were small and did not react to light, and there were small retinal hemorrhages, and on the third day the vision was only slightly blurred.

Beach and Cobb's case. Bulging of conjunctiva from extensive hemorrhages, pupils equal and reacted to light, no fundus lesions.

Sir George Beatson (7) in 1907 reported a case in which there were subconjunctival hemorrhages, proptosis oculi; ophthalmoscopic examination negative. Subsequent examination by Ramsey showed marked diminution in vision, beginning double optic atrophy in temporal sides of disks, contracted visual fields; the loss of vision was progressive. This is the first case in which this result was noted.

Rönne's case. Ophthalmoscopic examination on the second day showed the fundus normal; on the day following, the patient complained of fog before the left eye, and he could see the flame of a candle and count fingers; five days after the lesion he had no perception of light in the eye, and examination showed complete amaurosis, no pupillary reaction; arteries and veins were normal in calibre. In the right eye a small fresh hemorrhage was found on the temporal edge of the papilla; this bleeding had not existed at the two former

examinations. Two weeks after admission deep physiological excavations were found in both nerves, with a depth of three dioptres. The amaurosis of the left eye remained total.

In Lang's case the sight partly returned in one eye, leaving a partial atrophy of the nerve. In Moeser's case also partial atrophy was found. Braun and Wienecke attributed the cause of the nerve changes to an orbital hemorrhage. Rönne, after a careful analysis of all the cases recorded, maintains that only a hemorrhage in the optic nerve can produce these results, causing degeneration of the nerve fibres by interrupted circulation, or by pressure of the hematoma, such a hypothesis explaining the transitory as well as the lasting loss of sight. Two cases of Béal showed an ophthalmoscopic picture of embolism of the central artery, with retinal œdema, thin arteries, with tortuous and dilated veins, and retinal hemorrhages. The wide variation in the ophthalmoscopic pictures in the reported cases, and the time at which vision was reduced, would seem to indicate that it depended upon the location of the hemorrhage in the optic nerve. The sudden blindness, followed immediately by optic atrophy, with retinal hemorrhages, would indicate a large hematoma in the optic nerve, between the papilla and the entrance of the ophthalmic artery and exit of the vein.

I am indebted to Dr. Elliot, of the Presbyterian Hospital, for the privilege of studying and reporting this case.

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EARLY CHANGES OCCASIONED BY THE INVASION OF THE EYE BY THE CYSTICERCUS.

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Abridged Translation from the *Archiv f. Augenheilkunde* (the German Edition of these Archives), vol. lvii., No. 4, by Dr. Alfred Braun, New York.

(*With Appended Plate A.*)

MANY communications, besides my own, have appeared to show what changes are produced in the membranes of the eye by the presence of a cysticercus. However, these were observations made on eyes which had undergone extensive changes, sometimes atrophic, sometimes purulent, which were enucleated months and years after invasion of the eye by the parasite. None were based upon the early stages of the invasion of the vitreous by the cysticercus. It is clear that these histologic findings are not suited to show us which changes are really due to the action of the parasite, and which are due to the mere presence of the cysticercus acting as a foreign body.

This examination has also the practical value, that it aids in determining the factors which sometimes allow of a successful extraction of the cysticercus from the vitreous, and at other times give rise to such extensive alterations of the inner membranes of the eye that enucleation is necessary; it can also aid in explaining many clinical data, which appear in observations on cysticercus of the vitreous, and which, through want of anatomical control, are usually explained by more or less plausible hypotheses. Thus, for example, the usually rapid diminution in visual acuteness, sometimes

ending in light-perception, and often in complete blindness, does not appear to be explained by the ophthalmoscopic findings, which usually show us the media and retina in a condition in which we would expect to find a fair amount of vision. Furthermore, it is unknown why the cysticercus is sometimes movable in the vitreous, and sometimes fixed, or upon what anatomic condition those spots depend which are seen above the cysticercus, and which are perhaps more pathognomonic of the presence of the cysticercus in the vitreous, than the iridescent margin of the vesicle itself.

Fruitful in well-grounded deductions is only the study of the changes caused by the early stages of the invasion of the eye by the parasite. If we keep in mind the results of Leber's experiments upon the effects produced by the introduction of foreign bodies into the eye, we can easily distinguish between the changes caused by the cysticercus as a parasite, and those caused by the cysticercus as a foreign body; and keeping in mind the ophthalmoscopic findings, it will be easy to determine, in the microscopic serial sections, what the anatomical foundation of the various ophthalmoscopic changes is.

The following clinical observation is based upon a cysticercus which invaded the eye about 35 days ago, and the vitreous about 18 days ago.

B. Cal., a peasant, 49 years of age, noticed, while he was working, a feeling of heaviness in the left lid; two days later, he noticed that the left visual field was interrupted, and at the same time he noticed large red and green, sometimes round, sometimes netlike scintillating spots, which annoyed him very much. When he bent his head, the visual field of the left eye became very much clouded, and that of the other eye was also disturbed, so that he was compelled to give up his work.

He consulted an oculist in the neighborhood, who diagnosed a subretinal cysticercus. There followed intense pain, with perception of iridescent spots, with scintillation and distortion of objects, and the visual acuteness gradually diminished, so that on the 21st day after the beginning of the trouble, the patient was no longer able to distinguish one object from another. In this state, he came to see me, on January 2d, at which time I saw the following condition in the left eye:

Verlag v. J. F. Bergmann, Wiesbaden.

Kgl. Hofkunstanst. Bruckmann v. H. Stahl A.-G. München.

Externally, the eye shows nothing abnormal; the pupil is of normal size, but reacts very little to light. The media are perfectly clear.

The disk (Fig. 1) is covered by a whitish veil, which, gradually diminishing, spreads out in the surrounding retina for a distance of about two disk-diameters. It is almost on the same level as the retina. The opacity is greatest on the borders of the disk, and especially at its inner margin, where it is composed of white radial stripes, similar to those due to medullated nerve fibres, but not quite as shiny as these. They take in the inner third of the disk, its margin, and as much of the adjacent retina, and seem to overlie the diffuse grayish opacity. Corresponding to the exit of the vessels, *i. e.*, in the excavation, the disk is not covered by the diffuse opacity, but has an intense red coloration, so that it is almost impossible to distinguish the central vessels as they emerge. These vessels have irregular contours, and appear thin as compared to their real calibre, which is partly covered. They disappear under the opacity of the disk, to reappear and then disappear again, until at a distance of two disk-diameters, they continue the rest of their course uncovered. The arteries are slightly dilated, the veins turgid and varicose.

The retina is not only opaque, but shows, over an area of three disk-diameters, numerous radial, almost invisible folds. In the upper half of the retina, one can see a delicate grayish shading, which does not prevent one from recognizing clearly the outlines of the vessels, besides a dozen parallel short folds, which run from the inner side of the upper venous trunk. The most interesting changes are seen in the lower retinal quadrant, at a distance of about four disk-diameters from the disk. The circumpapillary grayish opacity stretches sideways and more downward, in the form of semitransparent milky brush-strokes, which cover the retinal vessels, sometimes entirely, and sometimes in part. The lower extremity of the opacity borders on a large, almost velvet-like spot, of a peculiar yellowish-white appearance, of about 2½ disk-diameters in extent, with two short brown stripes in the central portion. In this patch, there are two parts to be distinguished; the one consists of a rounded mass, about 1½ *mm* above the level of the retina, the other, more equatorial, but in contact with the former, which is not raised above the level of the retina, and shows a diffuse grayish color, cut through by very minute irregular folds of detachment.

A large vessel of dark red color separates these two portions from each other. This is the continuation of the lower retinal trunk, which, after a wavy course beneath

the grayish circumpapillary shading, reaches the patch, and after giving off a transverse branch, which goes inward, passes into the patch, where it disappears, to reappear again, and divide into two branches, an inner, which courses obliquely through the equatorial portion of the patch, beyond which it extends free, and an outer, which travels around the rounded portion of the patch, and then passes across to the outer quadrant of the retina. Both branches are of decidedly larger calibre on the patch than the trunk from which they take their origin, and in their circuitous course a portion of their calibre is hidden in the thickness of the patch.

To the outer side and forward, there is seen a cysticercus vesicle, which contrasts very strongly with the red of the background. It is transparent, of a beautiful sky-blue color, of a rounded, slightly flattened form, almost on a level with the retina. Only the peripheral zone is sky-blue, the central portion being whitish, and the intermediate zone pinkish-orange. In the central portion, there is seen a semilunar depression, with a smooth border, which surrounds the body of the parasite. This appears as a pear-shaped mass, with oblique axis, directed outward and upward, striated lengthwise, the larger end buried obliquely in the vesicle. From the small end, the neck of the parasite extends into the vitreous, rigid, and curved outward, making a very graceful and unforgettable ophthalmoscopic picture. The neck is less than two disk-diameters long, about half the diameter of the entire vesicle. It is flattened, and shows two raised borders, which give the impression as if the neck formed a flattened trough. There are no transverse markings. It is of a uniform gray color, which reminds one of the gray matter of the brain. The head is superimposed upon a constriction of the neck, and is rectangular; its diameter is only slightly larger than that of the neck. There can be seen with great clearness upon the head two suckers, and at certain moments, when the head is turned toward the observer, there can be seen four suckers, and the opening of the mouth. The hooks can be guessed at from their darker coloring, but one can distinguish nothing of their details.

Above the vesicle, and about ½ disk-diameter to the outer side, there are seen regular oval spots of various sizes, and in various directions, of a dark orange, almost rust color, somewhat paler at the edges. The largest are *2mm* long, and 1½ *mm* wide. There are about 50, but sometimes, when the parasite has changed its position, there seem to be less, returning again to the former number when it has returned to its original position.

The vesicle rests upon the retina with only a portion of its body, as the margins are raised, and there is a decided parallactic displacement of the retinal vessels. The margin of the vesicle shows an orange-colored *2mm*-wide areola, which is never seen simultaneously in the entire periphery of the vesicle, but disappears in that portion which is being looked at, to reappear in the remaining portion, and is always most marked in the portion opposite the head of the animal.

T. = n. V. = light perception below and outside, which in the two succeeding days disappeared.

The other eye is normal. The patient has never had worms in the stool, except round worms during childhood.

I took the patient into the clinic. Daily, every morning and evening, the fundus was examined, and it could be seen that the vesicle remained in the same position on the retina, but slowly changed its shape, from time to time. More than once, the head was found inverted, on which occasions the vesicle showed a sort of white shiny depression, surrounded by a grayish circular border. The continuous or induced electric current did not seem to be felt by the parasite; on the other hand, long-continued intense illumination aroused clearly defined lazy movements of the neck, similar to those of an elephant's trunk, and in the entire vesicle slight undulatory movements. Neither circulatory changes produced by pressure upon the eyeball and the carotid, nor the dropping of an eye-wash into the conjunctival sac had any effect upon the parasite.

I kept the patient in the clinic four days, and as he insisted upon radical treatment I did an enucleation. I came to this decision, because during the previous evening there occurred a marked retinal detachment at the site of the cysticercus, which would have made the result of an extraction doubtful.

Very instructive in this regard are those cases of cysticercus in the vitreous, in which extraction is done, in which after long-continued suffering it is necessary to do an enucleation; and not less instructive are the observations of De Wecker, who, taught by wide experience, wrote, in answer to Graefe of Halle, the chief exponent of extraction of intraocular cysticercus: "The operators who have followed the final outcome of extraction of cysticercus have found that in comparison with the immediate result, which is usually immediately published, it is not sufficiently pleasing to encourage one to undertake new operations." This statement is without doubt exaggerated, in so far as it relates to the

extraction of subretinal cysticercus, of which the end result is usually good, but is very proper in regard to extraction of cysticercus from the vitreous. A. Graefe, out of 45 operated cases, had 15 failures, which were almost all in cysticercus of the vitreous. Hirschberg, who has operated many cases of cysticercus, warns us to be very careful in judging the outcome of an extraction, as it often gives rise to unexpected and unpleasant surprises. The few cases, however, in which the extraction is followed by very good results make the statistics appear good, and encourage every clinician to operation, without which the eye is doomed. I would have prognosticated a very good outcome for my patient, as regards preservation of the eye, but not as regards restoration of the lost vision, if he had not refused extraction.

The globe was fixed for 48 hours, in 10% formalin. Before transferring it to alcohol, I cut away, with a sharp razor, a portion of the globe opposite the cysticercus, in order to observe its macroscopic appearance, which was very much facilitated by the formalin solution, which preserves the transparency of the tissues and the red coloration of the blood-corpuscles.

When the globe lies in its normal position, with the cornea in front, one notices that the cysticercus lies in the outer segment of the globe, a short distance behind the equator, attached to a protrusion of the retinal detachment; the head and neck no longer protrude; but the parasite shows itself as a round, opaque, smooth, milky vesicle; its margin is sharp, and the surrounding retina of normal appearance. In the lower part, which lies opposite the opening cut in the globe, the retina is not detached, but shows a white patch with irregular margins and surface, which measures about 3*mm* in its greatest diameter. On the inner side, the retina appears slightly wavy. There is no opacity in the vitreous, and no changes in the remaining portions of the eyeball.

I passed it through the usual processes, embedded it in paraffin, and stained it *in toto* with hæmatoxylin and eosin, and cut it perpendicularly to the sagittal axis, beginning at the optic nerve, and obtained, up to the portion where the cysticercus stops, 522 sections, each of 20μ thickness (alter-

nating with thinner ones). The cysticercus begins in the 250th section, and ends in the 322d section.

A large number I used for bacterial examinations and special stains.

The principal changes in the globe are of two kinds; the one may be described as diffuse, and the other local. To the first class belong:

1. The interstitial lymphoid infiltration of the optic nerve.
2. The perivascular lymphoid infiltration, especially of the large papillary vessels.
3. The preretinal cellular membrane.
4. The wrinkling of the three outer layers of the retina.
5. The destruction of the rods, and especially the cones.

To the second class belong:

1. The new formation of chorio-retinal cicatricial tissue at the point where the cysticercus forced its way from the choroid into the vitreous.
2. The atrophy of that portion of the retina underlying the cysticercus. These two last changes, as well as the shrinking of the outer layers of the retina, resulting in marked detachment of the entire lower-outer sector, were mechanical in nature, while the remaining changes were inflammatory.

The cysticercus, therefore, in addition to its mechanical effect, had an inflammatory action in this case, and inasmuch as the infiltration, the exudation, and the cellular new-formation were most marked on the surface of the retina and the disk, and to a lesser extent in their outer layers, and as the choroid and vitreous were not affected at all, we must conclude that the inflammatory effect of the parasite is developed centrifugally, and to only a moderate degree.

What the nature of this effect is, whether microbic or chemical, has been gone into very thoroughly. From the observation that in the further course of every eyeball containing a cysticercus, a plastic choroiditis, and often a diffuse or disseminate purulent choroiditis develops, some authors claim that the cysticercus, in passing from the gut to the eye, carries bacteria into the eye, which later develop here. Some authors even believe the cysticercus to favor the development of ocular tuberculosis. The bacteriologic

examinations, however, show that this is not the case. Leber was the first to make a culture in gelatin from a cysticercus extracted from an eye, with negative results, and to examine the intraocular fluids for micro-organisms, finding cocci in only one case.

In one case of Leber's, and in one each of Deutschmann and Treitel, micrococci were found, by the former in a culture prepared from the ocular contents, and by the latter in the tissues of the eye containing the cysticercus. Leber himself does not exclude the possibility of an accidental contamination of the culture; the other two cases were eyes in which extraction of the cysticercus was attempted, and in which the entrance of bacteria through the wound could not be excluded.

In my case, repeated examinations for bacteria by all the known methods were negative. Cultures and inoculations with the material from an eye extirpated for subretinal cysticercus with beginning painful phthisis bulbi were likewise negative. The cysticercus lay in a niche in the midst of a mass of new-formed cellular tissue, between the retina and sclera, around which there were observed, under the microscope, small areas of lymphocytes, like pustular miliary foci. Some material was carefully removed from the vesicle, and injected into the anterior chamber of the right eye of a guinea-pig, and some more into the vitreous of the left eye. After 24 hours, the right eye was irritated, the aqueous cloudy, and the iris dull. On the third day there was an intense inflammation. Some aqueous was drawn off from the anterior chamber with a hypodermic needle, and inoculated into two test-tubes, one with serum, and the other with glycerin-agar, but nothing developed. A similar negative result was obtained on the same media by inoculating with the material obtained from the vitreous of the left eye. In spite of the extensive injuries, both eyes healed, in one case with pupillary occlusion, and in the other with retinal detachment, which prevented a view of the eye-grounds.

Leber is of the opinion, with which I fully agree, that the cysticercus is amicrobic, but provides a favorable soil in the eye for the development of any bacteria that happen to be circulating in the blood, and the positive culture ob-

tained by him confirms this hypothesis, which also finds a substantiation in the examinations of Becker, Rosenbach, and Krause, who showed that the localization of the bacteria injected into the blood of the animals experimented upon usually occurred at a place where there was inflicted a marked trauma, such as fracture, *i. e.*, at a point where there was less resisting power.

We hope that, in the future, cultures will be made from the ocular fluids and blood in every case of ocular cysticercus.

As bacterial effects of the cysticercus are excluded, we must consider the irritative changes brought about in the eye by the cysticercus to be of a chemical nature. This does not occur with the same degree of intensity in the different tissues of the eye, nor does it last the same length of time in all cases. There is almost no effect in the tissues of the choroid, as even after the lapse of several months there were no changes to be seen in this membrane. There is very little effect on the vitreous, but a very marked effect on the retina and optic nerve, more marked in the superficial than the deep layers.

If we overlook the changes caused by the invasion of, and pressure upon, the retina by the parasite, the entire picture seems to indicate an irritating substance dissolved in the vitreous, which acts upon the tissues with which it comes into contact. This must stand in relation to the metabolism of the worm, especially its excretory products, which are seen in the living as a semi-soft, and in the hardened specimen as a coagulated, hyaline substance, which is found in the pericaudal space of every cysticercus, which I called the fecal-space of the cysticercus. In this case, such a substance is present only in that portion which lies in contact with the retina, this effete material being gradually absorbed in the vitreous and exercising its irritating function upon the surface of the optic nerve and the entire retina, with lesser intensity in the upper quadrants.

From the clinical observations noted in the literature in which the retina remained for a long time in a condition of moderate opaqueness, and sometimes spontaneously regained its transparency, from the observations in which the retina remained normal over a greater or lesser area, in spite of the fact that a cysticercus lodged in the vitreous for a

long time, and from those cases in which, after a successful extraction of the parasite (Leber, Seifert), the retina rapidly regained its transparency, we must conclude that the irritative action of the parasite is very slight, and of short duration, in contradiction to Wagenmann and many others, who think that the disorganization of the eye is exclusively the result of the chemical action of the cysticercus, an hypothesis which finds no support in the histology or biology of intraocular cysticercus. Wagenmann, for instance, assumes that the cysticercus elaborates:

1. A noxious material, in larger quantities as it grows older;

2. A greater concentration of its irritating products through longer residence in the eye;

3. A varying power of irritation in its various stages of development; and,

4. A diminution of its power of irritation during its inactivity.

On the contrary, the study of an eye disorganized by the presence of a cysticercus shows us that alteration of the structure of the interior of the eye depends upon the new-formation of masses of connective tissue underneath the retina, which is identical with that which is observed in an eye in which any foreign body is lodged. Without repeating the arguments and the clinical and histologic appearances which I mentioned in another work, I conclude now, just as I did then, that except for a moderate irritative effect upon the retina, which is temporary, the cysticercus has the same effect upon the ocular membranes that is displayed by any aseptic foreign body which cannot be absorbed.

Upon comparing the histologic with the ophthalmoscopic findings, we see that the changes in the disk are due entirely to a small-celled infiltration; where this was slight (lower part of the excavation), it allowed the ground substance to shine through, and the disk appeared red. Where it was thick (inner border), the disk and its edge appeared not only covered by a white veil, but there was also a delicate striation, and as the infiltration was superficial, it covered the vessels, and where it formed a cellular capsule over these, the vascular lumen appeared irregular, and interrupted here and there.

Near the papilla, the retina looked grayish, but not on account of the small-celled infiltration, or its own opaqueness, but rather as a result of a delicate connective-tissue membrane, which covers it like a very thin semi-transparent veil. This membrane is present in all observations of cysticercus in the vitreous in which it was possible to examine the fundus, as well as in many cases of subretinal cysticercus, in which there is covered, not only that portion of the retina overlying the vesicle (De Vincentiis, Salzmann), but also, though less frequently, the entire retina and the disk (Hirschberg).

If the cysticercus remains in the vitreous for a long time, without there occurring extensive intraocular changes, the retina regains its transparency in part, especially after successful extraction of the worm (Alfred Graefe, Leber, Hirschberg, Seifert). This is explained by the predominating cellular structure of the preretinal membrane, inasmuch as the lymphoid elements return into the circulation, and only a very small portion, namely the spindle-shaped and the fibro-cellular elements, remain, which become organized into very delicate connective-tissue fibres, which, as they are few, and very fine, do not interfere with the transparency of the retina. In the circumpapillary and papillary portions, however, where the lymphoid infiltration is not only intense, but occurs within the tissue, a larger portion of the elements is transformed into opaque connective tissue, as a result of which the cloudy appearance of the disk and its edges persists, even after successful extraction of the cysticercus.

The folds of the retina depend upon a slight detachment from the choroid, or rather from the tapetum nigrum. It begins with a slight plication of the three outer layers of the retina, and is continued by the formation of a thin layer of transudate, which also penetrates between the rods and cones, and causes them to become loose. The white appearance of the retinal scar depends upon the layer of non-vascular, dense cicatricial connective tissue, and the irregular appearance of its surface upon the irregularities of the preretinal membrane, which is thicker here than elsewhere. In other cases, attention was drawn to a pigmented patch of chorio-retinal atrophy, or to a white patch surrounded by pigment (Graefe, Seifert), or a bluish spot (Hirschberg). In my case, the pigment striations

were caused by remains of the retinal pigment-epithelium, the pigmented cells of the choroid being in no wise implicated.

In the ophthalmoscopic examination, special attention was drawn to the prevesicular rusty orange-colored spots. Von Graefe saw them in his very first case of cysticercus in the vitreous, but he considered them to belong to the retina (Figs. 2 and 3, *Arch. f. Ophth.*, vol. i., p. 1). To his pupil Liebreich is due the credit of having recognized their true character, and of having reproduced them in one of his famous drawings (*Atlas of Ophthalmoscopy*, Plate 7, Fig. 5). He considered them to be the results of bites of the vitreous tissue by the cysticercus, in the effort to obtain nourishment, and he set up the hypothesis, which later showed itself not to be in harmony with the clinical and experimental observations, these showing that injury of the vitreous left no ophthalmoscopic changes. An histologic examination of these spots was never attempted before mine. My examination showed that the spots consist of cell groups lying upon the inner surface of the hyaloid membrane. I saw no trace of break in this membrane, or of irregularity in the arrangement of the adjacent vitreous fibres, which could have made plausible the hypothesis that these subhyaloid nests were the result of cell proliferation at those spots where the cysticercus protruded its neck into the vitreous. On the contrary, I was able to see similar cell-nests at distant points, where, in the process of preparation, the hyaloid was separated from the retina, in which this causation could not be considered. They are small cell-proliferations, analogous to those which are observed on the posterior surface of the cornea in certain affections of the uveal tract. However, I have never seen them in any case of detachment, or in any other intraocular condition, and they stand, therefore, in direct relation to the presence of cysticercus in the vitreous, of which they are a pathognomonic sign.

Before closing this communication, I must add some observations on the visual function. This was completely lost in the various cases of cysticercus in the vitreous, in the course of a few months, as the result of retinal detachment; in a few cases, the patient was conscious of the time of entrance of the parasite into the vitreous, not so much on account of

the pain caused by the stretching and compression of the ciliary nerves, as on account of the distortion of objects, the flickering and colored vision, which accompanied it. Following these occurrences, there appears a scotoma in the field, sometimes negative and sometimes positive. At the same time the cysticercus throws a shadow upon another part of the background, which moves if the cysticercus is movable, and often follows the configuration of the cysticercus so closely that the patient can accurately describe the movements of the neck and of the entire vesicle. This lasts only a short time, as the integrity of the underlying rods and cones is soon lost. From my histologic findings, by comparing them with the ophthalmoscopic changes, we can easily follow the succession of events which results in their loss of function.

The cysticercus produces upon the surface of the retina a new-formed membrane, which is firmly attached at certain points, *i. e.*, where the vessels are, and which, contracting as a result of organization, causes a shrinkage of the inner layers of the retina, as these layers are mostly longitudinal, and somewhat elastic. The outer layers contract into very superficial folds, under which a fluid, partly exudate and partly transudate, appears, which soaks through the rods and cones, and interferes with their regular nutrition and function. The retina soon becomes detached from the tapetum nigrum as a result of a new collection of fluid under the outer layers of the retina, and there are formed those superficial folds of detachment which I pointed out in the region of the disk and near the cysticercus. In this way occurs the first stage of retinal detachment, which lasts for a varying time in the different cases, and which may partly retrogress, if the cysticercus is extracted from the eye. Usually, however, there follows an extensive and then a total detachment. It is not improbable that this sudden change in the ophthalmoscopic picture is due to perforation of the retina as a result of movements of the neck of the cysticercus, resulting, according to the investigations of Leber, in the fluid which lies between the hyaloid membrane and the caudal vesicle finding its way into the subretinal space; however, after the detachment is once started, the perforation is only necessary to explain the rapidity with which, in my case,

and in other cases of cysticercus of the vitreous, the detachment of a large portion of the retina occurred.

From this histologic examination, we can draw a practical lesson. The cysticercus lies, at least at the beginning, in many cases, under the hyaloid membrane, and not in the vitreous itself. An indication of this location is given by the prevesicular spots, which, as they lie under the hyaloid membrane, can be seen only with the ophthalmoscope, when the cysticercus is also situated under the hyaloid membrane. This sign is therefore valuable for the surgical treatment, as in this case the extraction of the cysticercus can be as simple as in the case of a subretinal cysticercus, on condition however that the incision does not open the hyaloid membrane, and falls exactly in the subretinal space, where the vesicle lies. Then the cysticercus passes out together with a greater or lesser quantity of lemon-yellow fluid, which surrounds it, and often without a trace of vitreous (Seifert, Reymond). This appearance has not escaped Leber, who explains it by assuming that the vitreous surrounding the cysticercus has become so dense that a large portion of the vitreous space is no longer filled with vitreous body, but with the fluid which flows out during the extraction. From our anatomical findings, however, we must conclude that in these cases the incision opened only the preretinal space, and did not involve the hyaloid membrane and vitreous.

CONCLUSIONS.

1. Cysticercus in the vitreous produces an irritating effect upon the retina and optic nerve, and gives rise to the formation of a preretinal cellular membrane, which is firmly attached to the retina at only a few points; upon this membrane and the small-celled infiltration, depends the cloudy appearance of the retina and disk.

2. The contraction of the preretinal membrane causes a contraction of the inner layers of the retina, and a throwing into folds of the three outer layers, and this causes the wavy appearance of the fundus, and starts the retinal detachment.

3. Cysticercus of the vitreous can be located beneath the hyaloid membrane, in which case there exists, between it and the parasite, a space, which is filled in by that lemon-

yellow fluid which flows out during extraction of the cysticercus, and which, when the parasite makes an opening through the retina, as a result of movements of its neck, may flow into the subretinal space, and give rise to an extensive detachment of the retina.

4. The small spots which lie in front of the cysticercus (prevesicular spots) depend upon the presence of subhyaloid cell-nests, and the iridescence of the margin of the vesicle is an optical phenomenon.

5. The white retinal patch at the point where the cysticercus forced its way into the vitreous is composed of compact cicatricial tissue, which causes a firm adhesion between the choroid and the remains of the retina, which is here represented by a few Mueller's fibres.

6. In the region where the parasite is attached to the retina, the latter is transformed into a connective-tissue membrane, and the cysticercus does not lie in direct contact with it, but through the intermediation of a more or less compact layer of cells and coagulated hyaline substance. Beyond the cysticercus, the retina rapidly takes on its normal layers again.

7. Numerous multinuclear bodies are observed on the surface of the preretinal membrane, near and underneath the vesicle. These are pseudo-giant-cells, which result from hyaline masses, in which nuclei have become entangled, and which take on the form of giant cells, probably as the result of the undulatory movements of the vesicle.

8. The extensive changes found in eyes enucleated late on account of cysticercus are the result of proliferation and change of the choroid, produced by the cysticercus, which acts like an aseptic foreign body, and not upon its irritative action, which does not affect the choroid.

9. The extraction of the cysticercus from the vitreous is favorable only when the prevesicular spots are seen, in which case we must be careful to reach the subhyaloid space, without opening the hyaloid membrane.

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A CONTRIBUTION TO THE STUDY OF TRAUMATIC ENOPHTHALMUS

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TRAUMATIC enophthalmus is one of the most interesting orbital affections on account of its obscure pathogenesis and its rarity. Even at the present time we are far from possessing a satisfactory theory of the mode of development which will suit all cases. This is explained by the numerous and complicated conditions which may all occasion or contribute to the same symptom of a recession of the globe into the orbit, and which may themselves be directly or indirectly due to the results of mechanical violence.

A study of the various hypotheses—there are upwards of nineteen, which may be divided into two main groups, according as they deal with mechanical or nervous factors—impresses one with the fact that no single explanation applies to all the cases. It would not be in accord with the actual conditions, were we, for the sake of a uniform point of view, to leave out of consideration any one of the many factors which are present and frequently, perhaps, co-operate. Lederer, for example, has attempted to prove by careful analysis of the cases heretofore published that an orbital fracture is invariably the underlying cause of the affection, yet his theory fails to satisfy the conditions in every case, even if we admit the possibility of a fracture of the orbital wall running its course without any other symptoms. The

cases of Manz, Schapringer, Grunert, Kilburn, and de Schweinitz cannot be explained in this way, and even more serious objections can be raised to the other theories which have been advanced.

It certainly seems a more scientific method to analyze carefully each case which comes under observation and to determine the factors which cause the enophthalmus, than to grope for a hypothesis which shall be applicable to the entirety of cases.

These were the considerations which led us in the interpretation of four cases, observed in the Leipsic University Eye Clinic, which are reported in detail below with a separate epicrisis for each case, and followed by an analysis of the most important points in the pathogenesis of traumatic enophthalmus in the light of a study of the literature.

CASE I.—This case was reported by Grunert in 1898. As the patient's statements varied considerably, the data which do not agree with the case-history or status as determined by us are put in brackets.

Phil L., aged 49. (Tuebingen, June 26, 1898, Leipsic, September 3, 1900.) In February, '97, the patient struck the back of his head in falling. [No] blood flowed from the nose and ears, and the left half of the head was occupied by a suggillation. The patient claims to have been unconscious for 4 days and to have vomited repeatedly. [Headache was said to have been constant for 14 days after the injury.] Soon after the fall, he began to notice at times an annoying buzzing and roaring in the left half of the head. After 3–4 weeks a small bluish red growth about the size of a lentil appeared at the nasal side of the left eyeball, and rapidly increased in size [grew slowly]. The eye protruded from its socket, and began to pulsate [vision being gradually lost], while the noises in the head increased, and severe headache came on which was increased on stooping [patient did not complain of headache or any other discomfort]. It is claimed that there was a knotty swelling on the brow above the left external angle of the eye. [The patient claims that a scar, now seen on the brow, is due to being struck with a brick.]

About 6 weeks after the accident, L. was admitted to a hospital in Carlsruhe, where, after 6 more weeks, the carotid was ligated and the phlebectasies treated with the galvano-cautery. About 3 weeks after operation it was noticed that the eye had sunk back into the orbit [patient

claims that the eye was sunken 3 weeks after the injury] and that the pulsations were weaker. After his discharge the patient went back to work for a time, but the buzzing noises and left-sided headache became worse again.

He also declares that vision had decreased in the right eye, complains of seeing as through a fog, and claims that light appears to him only as a globe with colored rings.

Physical Examination.—Three irregular linear scars on the scalp, under which infractions of the bone can be plainly felt. On the left side of the neck there is a cicatrix 15*cm* long, along the border of the sterno-cleido-mastoid muscle.

There is a striking enophthalmus of the left eye, the exophthalmometer measuring 14*mm* in the right, and 11*mm* in the left eye. The globe is displaced slightly downward and outward. The left lid fissure is wider open and can be closed only with great exertion. The orbital furrow is so deep, particularly above, that the finger can be carried into it for quite a distance. There is limitation of motility, especially outward. When the eye is palpated, pulsations are felt which are synchronous with the radial pulse ($\frac{2}{100}$ to $\frac{3}{100}$ seconds behind the carotid pulse) and become more marked when the eye is abducted. The ocular conjunctiva is raised up by a mass of tensely filled veins (between which some white linear scars are seen, which are said to have been caused by the electrolysis needle). On stooping, these veins become so distended that a nodular tumor is formed which protrudes from the palpebral fissure, the globe being dislocated outward to such an extent that the apex of the cornea reaches the orbital margin. Stooping also causes intense pain referred to the head and to the eye itself. On auscultation, no murmurs can be heard over the eye or in its vicinity (when the stethoscope is placed on the closed lids, a weak rhythmic murmur is heard).

O. S. There is a delicate opacity of the cornea. The pupil reacts promptly. (There is no direct, but good synergetic reaction; the media are clear.) The disk is perfectly white, with no changes whatsoever in the vessels. There is an excavation about 1.5*mm* deep.

V. O. Perception of light at 1*m* with uncertain projection. After the patient has kept his head lowered for some time, there is no perceptible dilatation of the retinal veins, and no visible arterial pulsation.

O. D. The anterior ciliary veins are slightly tortuous, not excessively full; the cornea is perfectly clear and transparent; the pupil reacts well. V. $=\frac{6}{12}$, unimproved. Retinoscopy shows emmetropia.

Ophthalmoscopical Examination.—Temporal half of the

disk quite atrophic. Marked glaucomatous excavation about 4D. deep. Upper nasal contraction of the visual field.

To determine whether the pulsation was transmitted to the cerebro-spinal fluid, lumbar puncture was performed on September 6, 1900, with a determination of the pressure which showed that the latter was not increased and that there was no pulsation. Pilocarpin was administered as the patient continued to complain of cloudy vision and colored halos.

September 25th. After ligating a number of markedly dilated afferent vessels, the epibulbar venous plexus was dissected off. A cavity was revealed in free communication with the cavernous sinus. Tampon and pressure bandage were applied. During the next few days, the patient complained of painful pressure in the orbit, loss of feeling in the skin of the left side of the head, and lack of appetite. Frequent vomiting took place. Temperature, normal; pulse full and regular. Dressings were changed every two or three days; the wound cavity looked well, bled but slightly, and began to granulate from the depths. The globe does not pulsate. The patient's complaints continue and there are psychical disturbances which become so marked that on November 6th the patient attempted suicide by hanging, necessitating his removal to an insane asylum. After his return, pulsations again became noticeable. At the nasal side of the globe lymph spaces are seen full of blood which are easily emptied by pressure but immediately fill up again when it is removed. The upper lid covers the globe almost completely and can hardly be moved.

January 26, 1901. The exophthalmometer registers: right, 15*mm*; and left, 10*mm*. Sensibility has returned on the left side of the head, as far as the hair border. V. O. D. $=\frac{4}{11}$.

After about 3 months spent in the asylum, L. again came to the eye clinic on April 17th. Status, about as before. O. S. slight rhythmic pulsation, anæsthesia of the left half of the brow; temporal dislocation of the globe; blood-filled lymph-spaces on the nasal half of the conjunctiva bulbi. The buzzing in the left side of the head has disappeared. O. D. V. $=\frac{6}{6}$, with complaint of cloudy vision. Visual field scarcely contracted. The left eye appears to be bound to the upper and posterior part of the orbit by broad cicatricial bands, so that it stands higher than the right. On passive raising of the upper lid, the globe rotates upward. The recession has increased to such an extent that it can no longer be measured with the

Sattler exophthalmometer. The cornea is intact, the refracting media clear. There is complete amaurosis, with the ophthalmoscopic picture of optic nerve atrophy.

On March 21, 1905, L. again presented himself, with the old complaint. O. D. V. = $\frac{6}{10}$ –. No improvement. With +3D., sph., Jaeg. No. 4–5, some words, at reading distance. T. somewhat increased. Pupil very small, reacts well. Cornea smooth and clear. Visual field contracted upward and inward. Nerve head, excavated. O. S.: Amaurosis. Disk, atrophic. An intended iridectomy is not done, on account of a bilateral purulent otitis for which the patient is transferred to the ear clinic, and discharged on March 25, 1905, with pilocarpin, 1%, and sph. +3. 5 D. O. D. for reading.

The symptoms in this case can be explained by the assumption of a fracture of the base leading to an arterio-venous aneurysm. L. was unconscious for 4 days, blood flowed from the nose and ears, there was a marked ecchymosis of the left side of the head; all signs of cranial fracture. Then, in the course of several weeks, a small bluish-red tumor appears at the inner angle and the eye begins to pulsate and protrudes. With this there are roaring noises in the left side of the head. Later, dilated veins appear beneath the conjunctiva. We have the picture of a pulsating exophthalmus, so often described, and due, as Sattler has shown, to an arterio-venous aneurysm in the cavernous sinus.

An especially interesting feature of the case is the subsequent development of enophthalmus, an unusual occurrence, indeed. This case seemed, if any did, due to scar formation in the orbit. The enophthalmus appeared about three weeks after the ligation of the carotid and cauterization of the phlebectasies. We may, accordingly, infer that these operative procedures bore a causal relation to the morbid process. As ligation for pulsating exophthalmus does not usually lead to recession of the globe, we can hardly attribute the development of enophthalmus to this procedure. The extent of possible destruction with scar formation in the orbit caused by the galvano-cautery could not be determined, as a later operation, extirpation of the venous plexus, still further modified the conditions. It is possible that matters were further complicated by the presence, in addition to

the factors causing exophthalmus, of others such as fracture of the orbital wall, blood extravasate, atrophy of the orbital fat, which are known to lead to traumatic enophthalmus. The clinical picture was, however, dominated by the signs of pulsating exophthalmus, and it was only after this had been cured by the ligation of the carotid that the other complications appeared. Whatever may have been the factors contributing to the enophthalmus, there is no doubt that a certain rôle was played by cicatricial bands which anchored the globe to the posterior and upper part of the orbit, while in the lower part of the socket an unusual depth of the conjunctival sac indicated the loss of the tissues usually filling out the floor of this cavity. The scar formation is to be attributed partly to the cauterization, and partly to the extirpation of the venous plexus, later on. The gradual increase of the enophthalmus, shown by instrumental measurement, can easily be explained by further shrinkage of the scar tissue. This does not exclude the possibility of other mechanical agencies, such as hemorrhage, atrophy of the orbital fat, and fracture of the wall of the orbit with hernia of the soft parts toward or into an accessory cavity. On the contrary, the sudden development of amaurosis speaks for a fracture involving the wall of the optic canal, and leading to injury of the trunk of the nerve, either by a bone splinter, or by a hemorrhage into the nerve sheath.

CASE 2.—January 12, 1903, Rich. N., aged 15, was struck in the right eye by a broom thrown from a distance of about 6*m*. He does not know whether he was hit by the broom-stick or the twigs. There was free bleeding and much swelling, so that the eye could not be opened. A skin wound of the upper lid was stitched, and healed by first intent. At the end of 8 days, the patient was able to open his eyes, but sight was completely lost.

Physical Examination.—Linear scar, slightly convex upward with partly thickened edges just below but not adherent to the upper orbital margin. No tenderness in this region. The right palpebral fissure is narrowed, measuring at the middle 4*mm*, as against 8*mm* in the left eye. The upper lid covers about ⅓ of the cornea. The lower lid is slightly swollen, and covers 2*mm* of the cornea, as against 1*mm* in the left eye. There is some bluish discoloration, and a small, flat thickening, of cartilaginous

consistency, can be felt through the skin of the lid which is gradually lost above, and can be sharply defined below. The conjunctiva is much injected and swollen and protrudes in thick rolls. The finger cannot be inserted between the globe and the wall of the orbit, meeting a rather decided but elastic resistance. The right eye lies 2.5*mm* deeper than the left and is turned slightly upward. There is no irritation. Motility is markedly limited in all directions and completely abolished downward. The cornea is smooth and transparent; the pupil widely dilated, somewhat oval with the axis upward and inward. The iris is 3*mm* broad above and outward, 1*mm* broad below and inward. The media are clear. There are no tears of the sphincter margin.

Ophthalmoscopic Examination.—Nerve head, white. Numerous old hemorrhagic deposits in the region of the macula, between which some glistening white and black strands appear. A month later, the enophthalmus had increased, measuring 3.5*mm*. The gristly strand beneath the lower lid can now be felt only as a diffuse thickening. The finger can be carried deeper between the globe and the orbital wall. The cornea is only about half covered by the upper lid. Motility has increased slightly. The pupil is still absolutely rigid, but decidedly smaller, the greatest breadth of the iris upward and outward, being 5*mm*, the least, below, about 2.5*mm*. The macular hemorrhages have disappeared; there are only slight traces of the white striæ. Eserin produces a contraction of the pupil, ad minimum. H. 1.5 D., under atropin. After about two months more, the globe is found to be somewhat more mobile, the most marked limitation of motility being toward the temporal side. Three months later the following condition was noted:

Right lid fissure, 2*mm* narrower than the left (7:9), the upper lid hangs lower, the lower lid is higher, than in the left eye. The right eye lies deeper and decidedly higher, 2–3*mm*, than the left. On forced upward rotation of the left eye, the right turns up only ½*mm;* on maximum downward rotation of the left, the right moves down only 2*mm*. On abduction the right eye lags only 3*mm* behind the normal excursion, and but very little behind on adduction. The right pupil is 7–8*mm* in diameter and rigid. The nerve head is snow white, the arteries extremely fine. In the region of the macula there are a number of glistening, white points, the fovea being apparently occupied by a somewhat larger white spot. V=0.

First, in regard to the nature of the injury in the case

presented, it may well be assumed that the eye was struck by the handle of the broom and not by the brush. The trauma acted with considerable force on the region of the right upper orbital margin, producing a wound of the upper lid. The profuse hemorrhage and the position of the scar show that the orbital margin itself was struck in the vicinity of the frontal artery, but there were also signs of direct injury of the lower orbital margin, namely, the cartilaginous thickening which afterwards receded. Although no symptoms of basal fracture appeared, there is a very strong presumption that a fracture of the orbital wall took place which extended into the optic canal and injured the nerve at this point, producing optic atrophy leading to amaurosis. The pupillary paralysis and the retinal hemorrhages show that there was, besides, an injury of the globe itself. In this case too, accordingly, a fracture of the orbital wall must be assumed as the cause of the enophthalmus.

The thickening felt on palpation below the upper orbital margin and the rather firm resistance below the globe must be interpreted as subperiosteal blood extravasate. The enophthalmus came on rather soon after the injury, as in Case 1. Within three weeks it was quite decided (2.5*mm*) and within a month more amounted to 3.5*mm*. This increase must, again, be attributed to cicatricial contraction of connective tissue, newly formed in place of the orbital tissues which had been destroyed. Cicatricial bands also explain, at least in part, the limitation of motility which was particularly marked on abduction and upward rotation. The ptosis, which gradually improved although the enophthalmus increased, must probably be attributed to a direct lesion, probably hemorrhage, of the levator muscle. An involvement of the sympathetic can neither be affirmed nor denied, as the behavior of the pupil and of the lid fissure throw no light on this point. The development of the enophthalmus can certainly not be ascribed to a lesion of the sympathetic alone, as there were other circumstances which spoke in favor of a mechanical cause. While the chief rôle was played in this case also by a fracture of the orbital wall, we cannot exclude the mechanical action which the suddenly rebounding eyeball exerted upon the orbital fat tissue.

CASE 3.—Anna F., domestic, aged 16. January 19, 1903. On December 5, 1902, the patient had been kicked by a horse on the right temple. On examination, two days later, a doughy swelling was found at this point, reaching from the zygoma to the ear, and involving the lids. The anterior chamber was filled with blood, so that the interior of the eye could not be seen. The eye was painful on motion only. It was stated that the hemorrhage had not appeared until the day before. V = 0. Atropin and warm compresses were ordered. After two days, the blood in the anterior chamber had become absorbed, but there were large masses of blood in the vitreous so that no fundus reflex could be obtained. Several days later the patient was admitted to a hospital. There was a second slight hemorrhage into the anterior chamber which quickly disappeared. The doughy swelling increased and broke spontaneously, discharging a large quantity of thin, dark blood. The wound soon healed. The hemorrhage in the vitreous cleared up somewhat so that a red reflex appeared in the depth which on focal illumination appeared as broad light-red plates. The patient could count fingers outward toward the right. The swelling of the soft parts became less and less, and a flattening of the lower orbital margin more and more, noticeable. There was never any tenderness. On January 12, 1903, a third hemorrhage took place into the anterior chamber. The possibility of a wound of the posterior lower segment of the globe, possibly by a splinter of bone, was thought of, and permission for an operation asked, and refused. The bleeding became more copious and the patient was transferred to the eye-clinic.

Physical Examination.—A sharp edge is felt, corresponding to the inferior orbital incisure. From this point on to the zygoma, the lower orbital margin is crushed in. About 1*cm* below the external canthus the skin is adherent to the bone to a point beyond the zygoma. The hollow between the upper lid and the orbital ridge is deepened, and the lower lid is flatter than the left. The globe lies decidedly deeper than normal, and is freely movable except for a slight limitation of abduction. The cornea is unaltered. The anterior chamber is somewhat deep. There is a hyphæma about 2*mm* deep. The pupil is drawn outward and upward. No consensual reaction of the left pupil can be elicited. Blood in the vitreous prevents ophthalmoscopic examination. Functional examination shows perception of a candle flame at 2*m* with uncertain projection. Tension somewhat less than in the left eye, increased somewhat in the course of the next few days. No pain. O. S. V. = $\frac{1}{3}$. Accepts Sph. +0.5 D.

This case shows, like those which have preceded, that a severe injury to the margin of the orbit was followed by fracture of the orbital wall. Two weeks after the injury a decided enophthalmus develops. It is hardly to be assumed that this symptom was present two days after the injury, at the first examination, as it could hardly have escaped the notice of the examining physician, particularly as no lid swelling of any account had yet developed, which could have prevented examination of the globe. We must infer, then, that a certain time elapsed before the picture of enophthalmus was presented.

In this case again the globe itself presented evidence of injury by blunt violence, in the form of hemorrhages into the anterior chamber and the vitreous. Whether there was also a detachment of the retina, or whether the nerve was injured by a fracture extending into the optic canal, cannot be decided, as no examination of the fundus could be made. At all events there was in this case no amaurosis immediately after the injury, such as was observed in the other two cases. The deformity of the lower orbital margin makes it more probable even than in the other two cases, in which a fracture of the orbital wall was assumed, also, that there was an infraction of the lower wall of the orbit into the maxillary antrum, allowing the soft tissues to give way in this direction. It cannot be denied that a number of other mechanical factors may have been active in this, as in other cases. Among them we may mention, atrophy of the orbital fat by pressure from the globe, from hemorrhage, from pressure or traction by the fractured bones; secondary scar formation in the disorganized orbital tissue, fixing the globe, and in contracting increasing the degree of enophthalmus. The real cause of the enophthalmus is most logically sought, however, in the fracture of the lower wall of the orbit.

CASE 4.—Otto Sch., trooper, aged 21. On February 1, 1905, the patient was kicked by a horse in the region of the inner angle of the right eye. Vomiting, epistaxis, and partial loss of consciousness, with marked swelling of the lids, were noted. After the swelling had gone down, Sch. noticed that the right eye was sunken and that the vision was bad. The displacement of the globe and the disturbance of sight became worse within a short time.

Physical Examination.—February 13th. O. D. lies much deeper in the orbit than the left eye. The exophthalmometer shows a difference of 2.5*mm*. No limitation of motility. There is a subcutaneous abscess at the right inner canthus. The palpebral fissure is narrowed. In the region of the floor of the frontal sinus there is a broad prominence which is tender, as is also the region of the inner margin of the orbit. The root of the nose is displaced to the left. A skiagram shows very clearly a depressed fracture of the lower wall of the orbit extending some distance downward from the margin. Skiascopy shows As. M.—1D. axis 180°, O. D. O. S., E. V. O. D.= $\frac{6}{18}$. V. O. S.= $\frac{6}{5}$.

Ophthalmoscopic Examination.—Negative.

O. S. Small central scotoma, for color only. Some disturbance of red-green perception. No diplopia. Decided divergence of the visual axes.

This case shows a marked similarity to the preceding both in the nature of the injury (blow from a hoof) and in the symptoms. Here a fracture of the lower margin of the orbit was found, which, as shown by the X-ray, involved the lower wall. Skiagraphy may be a welcome aid in the elucidation of the pathogenesis of traumatic enophthalmus, the more so as fracture of the orbital margin may be diagnosticated clinically from its deformity, but not one of the orbital wall.

In this case, again, the dislocation of the globe appeared soon after the injury. As soon as the swelling of the lids had gone down, the patient noticed that his right eye lay deeper than the left. The behavior of the eyeball is particularly interesting. In marked contrast to the almost complete loss of sight in the other three cases, vision was only reduced to $\frac{6}{15}$. As the fundus was found to be perfectly normal, even this slight disturbance of sight seemed hard to explain. Tests with colored objects then revealed a small relative central scotoma. The case resembles one reported by Fuchs, in the last edition of his text-book (1905), of a man of 40, who had a slight lesion of the optic nerve due to chronic inflammation of the posterior ethmoidal cells and sphenoidal sinus. The only symptom was a small central scotoma—for red only. A case with similar etiology and symptomatology was recently observed by Birch-Hirschfeld. These observations suggest the presence in our case of a slight lesion of the

nerve, either from traction or from a hemorrhage into the sheath. The clinical picture was dominated by the fracture of the lower wall of the orbit, so that it seems logical to attribute the development of the enophthalmus to this factor. There was no evidence of a lesion of the sympathetic, at least at the time the patient was examined. The pupils were equal, and reacted normally; the tension was the same in both eyes.

A review of the four cases reported shows a correspondence as to certain features. In all four, the dislocation of the globe followed a severe injury which had involved the orbital margin and produced a fracture of the wall of the orbit. In the first two cases this complication was made probable by the development of amaurosis with the picture of optic atrophy, and actually proven in the others by the deformation of the orbital margin and of the floor of the orbit, respectively, as shown by the X-ray.

The first case was complicated by the symptom complex of traumatic exophthalmus which was the feature at the beginning. In the other cases the enophthalmus became pronounced a few days or weeks after the injury, and increased later, apparently on account of scar contraction or atrophy of the orbital fat. A uniform interpretation of the pathogenesis of traumatic enophthalmus can hardly be given on the basis of 4 cases. It would be natural to accept Lederer's explanation and to view the orbital fracture common to all four cases as the principal cause. It seems more logical, however, to base our judgment on a careful analysis of all previous observations. Lederer's statistics which have been brought up to 1905 by Meltzer give a complete survey of the literature. With the four cases from the Leipsic Eye Clinic detailed above, these tables comprise 63 cases of traumatic enophthalmus. Seven cases, those of de Schweinitz, Kilburn, Morton, Ogilvy, Hansell, Tweedy, and Causé, are not included. For the sake of completeness a short résumé of these cases is given here.

CASE 64.—De Schweinitz. Male, aged 23. Struck against a wagon. Partially unconscious for 4 days. Lids swollen. Diplopia. Left eye sunken; no fracture made out. After 11 days, O. S. enophthalmus of

4*mm.*, lid fissure narrow. Motility limited upward-inward and up and out. Interpreted as a lesion of the sympathetic "as there was no evidence of fracture of the orbital wall."

CASE 65.—Kilburn. Male, aged 53. Fall from a sled. Left upper lid injured by a spectacle glass. After 15 days, enophthalmus. Skin scars, slight ptosis, lid fissure much narrowed. Fundus normal. Interpreted as a tear of Tenon's capsule and the tarso-orbital fascia, with possible peripheral paralysis of the sympathetic. The time was too short for any agency of scar contraction.

CASE 66. Tweedy. Thrust with a cow's horn. Dislocation of the globe into the antrum (analogous to Becker's case). Amaurosis. Enucleation.

CASE 67.—Morton. Male, aged 40. Dragged by a cart. Unconscious. Bloody suffusion of the eyes. Superior maxilla crushed in. Bilateral enophthalmus, more marked in the left eye. Pupils irregular and rigid (posterior synechiæ). Motility limited in all directions. Detachment of the retina. Tension lowered.

CASE 68.—Ogilvy. Male. Trampled on by a steer six months before. Left globe dislocated inwards and downwards. Three fractures of the orbital wall, one at the outer third of the upper margin, one at the outer angle, one at the maxillary border. Loss of motility. Convergent squint. Rupture of the globe. Amaurosis. Interpreted as due to fracture.

CASE 69.—(Cf. De Schweinitz.) Hansell. Blow on the orbital margin. Enophthalmus noted immediately after the injury.

CASE 70.—Causé. Male, aged 46. Kicked by a horse in the left eye. Unconsciousness for several hours. Swelling of the lids. After disappearance of swelling, eight days later, enophthalmus noted. V = $\frac{6}{6}$. Fundus normal. Motility downward, more extensive than in the right eye. Left lid fissure narrowed, dilating with the pupil after cocain. "The sympathetic thus shows normal functions so that it may be excluded as an ætiologic factor in the interpretation of the clinical picture." Cause, fracture or infraction of the lower orbital wall with callous thickening.

A study of the 70 cases cited shows that there is no uniform pathogenesis. The old theory of Himly (breaking off of the trochlea) need hardly be discussed, as Beer and Lederer have already disproved it, and hardly a single author now supports it.

Gessner's view, on the other hand, that enophthalmus is caused by scar contraction in the retrobulbar tissues, does

not seem unjustifiable. Our first case supports it, as does Causé, although it certainly does not fit the cases in which enophthalmus developed much too soon to be explicable by retrobulbar scar shrinkage. The hypothesis of Nieden (atrophy of the retrobulbar tissue by pressure backward of the eyeball) seems to apply to only a few cases, while the majority of the observations speak against it. Atrophy of the fat tissue undoubtedly must be taken into consideration in a number of cases of traumatic enophthalmus, but it is usually combined with lacerations and hemorrhages in the retrobulbar tissue, or due to lesion of the sympathetic, and is not to be attributed simply to temporary compression.

Lederer's view that in most instances fracture of the orbit plays the principal rôle is supported by our observations, and still more by a survey of the entire series of reported cases, but even here this theory is not completely satisfactory, as it does not fit the cases in which enophthalmus developed soon after the injury.

The fracture, per se, does not explain the development of an enophthalmus. Either it leads to hemorrhage, and this again to the formation of retracting scars, in which case we should expect exophthalmus, leading, much later, to recession of the globe into the orbit; or the fracture leads to enophthalmus indirectly by causing a depression of the orbital wall, particularly the floor, thus increasing the retrobulbar space. In the cases of Ogilvy, Hansell, Morton, Lange, and others, as in our last three cases, this mode of development may be considered, to say nothing of the observations of Becker, Lang, Langenbeck, and Tweedy in which the globe was visibly dislocated toward the antrum. A depressed fracture of this sort would also explain the early development of the enophthalmus, but it must be admitted, on the other hand, that in many cases there is no clinical basis whatever for such an assumption.

Some of the authors explain the development of enophthalmus by a contusion of the orbital margin with laceration or tearing off of the fasciæ and ligamentous slips fastening the globe to the margin of the orbit. This is generally accompanied by an infraction or fracture of the margin or wall of the orbit, and complicated by a retrobulbar hemorrhage which

is usually absorbed without further deleterious consequences for the eye, but in isolated cases leads to the changes already considered by Lederer, particularly when other retrobulbar elements of the orbital contents were injured simultaneously. This explanation of Causé combines the causal factors of a number of other hypotheses to fit all cases, but I believe we can go still farther.

The neuropathic hypothesis warmly supported by Beer, Talko, Schapringer, and others cannot be dismissed lightly, as is shown by a careful study of the literature. A number of cases are recorded which prove that enophthalmus may develop in the course of neurotic facial atrophy. In many cases of traumatic enophthalmus there were symptoms which spoke in favor of an injury of the fibres of the sympathetic and the experimental investigations of Edmunds and Lawford give further support to this view.

As far as the previously reported cases show, the idea of traumatic enophthalmus embraces various determining causes. It is not easy to define the agency of each factor. It is possible that the X-ray may aid us in detecting a depressed fracture of the orbit which otherwise would escape notice.

Having gained a knowledge of the various hypotheses as to the cause of traumatic enophthalmus, new cases should be analyzed from every point of view. More careful observation will probably show that the clinical picture under discussion may be produced by various agencies, and not seldom, perhaps, by the combination of a number of mechanical or nervous factors.

COMPARATIVE STUDIES CONCERNING THE INFLUENCE OF THE ACCOMMODATION ON THE OCULAR TENSION IN VERTEBRATES.

By Prof. CARL HESS.

Abridged Translation from *Arch. f. Augenheilkunde*, the German Edition of these Archives, lxiii., No. 1, 1909, by Dr. Matthias Lanckton Foster.

(*With two text-cuts.*)

AFTER our studies had shown that the intrinsic muscles of the eyes of birds and mammals remained capable of electric stimulation for half an hour or more after enucleation, I instituted a series of comparative observations on the enucleated eyes of different vertebrates. For manometry I used Hering's micromanometer, which consists of a fine canula in which is cemented a short capillary tube closed above; in the closed end of the latter is a little bubble of air, while the rest of the tube, as well as the canula, is filled with physiological salt solution. After introduction of the canula into the eye very slight fluctuations of intraocular tension can be easily recognized through changes of the meniscus of fluid.

The management of the experiment is very simple: The enucleated eye freed from the attached muscles is placed on suitably bent needle electrodes, after the canula has been introduced into the anterior chamber or vitreous and fixed in the desired position; the meniscus in the capillary tube is observed by means of a microscope. Thus it is possible to follow the tension even in eyes of only 4*mm* axial length. In small reptiles the tension in the vitreous was observed; in birds that in the vitreous was taken in some, that in the anterior chamber in others. Among reptiles I have used the alligator, lizard, adder, and turtle for experiment. In all

there was a quick rise of intraocular pressure at the instant of stimulation, which returned rather quickly to normal when the stimulation was discontinued. The fluctuations in tension take place at almost the same time as the accommodative changes, which can be followed simultaneously by a second observer. Likewise in the enucleated eyes of birds every excitation caused an increase of the intraocular pressure.

Five monkeys' eyes and three dogs' eyes were tested in exactly the same way as the eyes of reptiles and birds immediately after enucleation, but in no case was the slightest change of the ocular tension perceptible on excitation, even with the strongest current, although weaker currents induced a lively contraction of the pupils and the manometer was so sensitive that gently touching the eye under examination caused a distinct change of the fluid meniscus.

Conclusions cannot be drawn that the behavior of the eye in question would be the same in the body and under normal conditions of circulation from the results obtained in the enucleated eye alone. It would be conceivable, for instance, that contrivances were present in the eyes of reptiles and birds which would suppress the sudden increase of tension caused by the rapid contraction of the muscles. But if we take into consideration how much higher the tension of the eye is under normal conditions in the body than it is after enucleation it is at least just as conceivable that the accommodative increase of tension is even greater in the former than in the latter, in which, in spite of the low tension the contraction of the intrinsic muscles produced a marked increase. At any rate it can no longer be assumed that the accommodation has no influence on the intraocular tension in the eyes of birds.

The fact is established that the effect of the contraction of the intrinsic muscles upon the ocular tension is altogether different in the enucleated eyes of birds and reptiles from that I have obtained thus far in mammalian eyes.

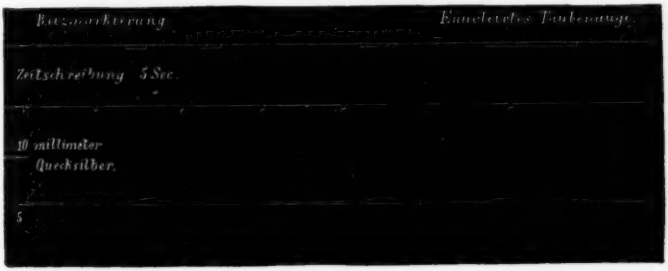
It is desirable that the results obtained should be measured and delineated graphically, and for that purpose Dr. Wessely has been so good as to place his apparatus for measurement and registration of minute fluctuations in the ocular tension with simultaneous registration of the blood pressure at our

disposal and to undertake the following very fatiguing experiments.

Curves 1 and 2, taken with Wessely's apparatus, show the ocular tension in a pigeon's eye with stimulations of different strength and duration; the eyes of other kinds of birds behaved the same.

The enucleated eye from which Curve 1 was taken had a tension of about *7mm;* a millimetre in height corresponds to a difference in the ocular tension of about 0.5*mm;* with the first stimulations the increase of tension was thus about 0.5*mm*, with the later ones somewhat less.

In the eye from which Curve 2 was taken the tension was about 11*mm* and increased with the first stimulations about



Curve 1

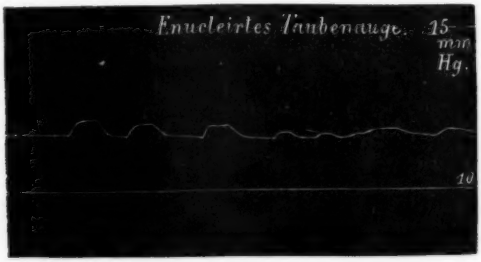
0.5*mm*. The fact that in such curves increases of tension of less than 0.25*mm* can be recognized with perfect distinctness gives an idea of the great sensitiveness of this method.

Repeated observations made with every precaution in the same manner on freshly enucleated monkeys' eyes again revealed no influence on the ocular tension. Currents of various strengths were employed, particularly such as induced the maximum myosis at the moment when the current was closed. As the register showed only a straight line it is not reproduced.

Another series of measurements were made with Wessely's apparatus on living monkeys after they had been curarized. Thus only could be excluded the involuntary contractions of the extrinsic eye muscles, which otherwise render it impossible to determine the influence of the intrinsic muscles

on the ocular tension. But on account of the great liability of the blood pressure of such curarized animals unexceptionable measurements of the ocular tension are possible only when the tension of both eyes or the blood pressure itself is registered simultaneously with the stimulation of one eye alone.

One eye of an animal was stimulated while canulæ introduced into both anterior chambers registered the tension in the two eyes; each time the tension fell uniformly in both eyes and rose again after the stimulation was discontinued. No difference in the tension of the stimulated and non-



Curve 2

stimulated eyes could be detected. The change in tension in both eyes showed that it was not due to the contraction of the intrinsic muscles, but to a change in the blood pressure. If the electric current was so weak that the ocular tension was no longer reduced in both eyes, no contraction of the pupil could be perceived, so that an isolated effect upon the intrinsic muscles of the eye could not be produced in this way.

In a second monkey examined in the same way the stimulation of one eye caused a slight, not noticeably different, increase of tension in both; this is easily explained by the fact that the blood pressure of such animals may be noticeably increased by slight sensory irritations.

The direct proof of the dependence of these changes of ocular tension upon the blood pressure is furnished by the following experiment. The tension in both anterior chambers and the blood pressure in the femoral artery were registered in a curarized monkey; at each excitation of one eye the tension in the two eyes fell nearly uniformly and in nearly the same

way as the blood pressure, the curve of which showed the slowing of the pulse characteristic of stimulation of the vagus.

We thus twice met with the fact that excitation with such electric currents as can produce contraction of the pupil in monkeys caused a distinct reduction of the general blood pressure. Whether this is the normal behavior, or an exception accidentally met with twice, is uncertain. The slight increase of the blood pressure met with in the second monkey mentioned corresponds more to what, with our present knowledge, we would have expected; any extraordinary liability of the blood pressure to sensory stimuli shows itself in such circumstances only in curarized animals. But any noteworthy increase of tension from accommodation must have been brought out by the alternate excitation of first one and then the other eye.

The results of these experiments agree with the results of former observations in showing that even with the most sensitive methods not the slightest increase of ocular tension can be perceived associated with strong contraction of the intrinsic muscles of the eye of the monkey or dog. Whether this is true for all mammals or not must be determined by further experiments.

It was settled by frequently repeated experiment that the play of the pupil caused by changes of light had no influence upon the ocular tension.

The fact that demonstrable accommodative increase of tension was absent in the eye of the dog could not be considered proof so long as the objection was possible that this absence in such weakly accommodating eyes did not include absence in eyes with much greater range of accommodation. But this objection is not tenable, for we have proven accommodative increase of tension in the eyes of night birds, whose accommodation is scarcely greater than that of young dogs, and the range of accommodation in the eyes of monkeys, which show no increase of tension, is three times as great as that of night birds.

It is an interesting question whether the accommodative increase of tension is met with in vertebrates only among the reptiles and birds, but my experiments on amphibia and fishes, as well as on other mammals, are still too few to enable me

to give a satisfactory answer. In various kinds of reptiles excitation with weak electric currents is followed by a distinct deformation of the unopened eye, in the snake's eye the existence of preformed scleral folds deepened by the accommodation can be demonstrated. This fact gives a satisfactory explanation of the accommodative increase of tension in the reptile's eye with demonstrable accommodative deformation of the coats of the eye. Whether the same explanation may apply to the eyes of birds, or whether other factors must be taken into consideration, remains uncertain at the present time.

COMPARATIVE STUDIES OF MIOSIS DUE TO LIGHT AND TO CONVERGENCE, AND OF THE ACTION OF ATROPIN AND ESERIN UPON THE IRIS AND CILIARY MUSCLE, TOGETHER WITH REMARKS CONCERNING THE FORM OF THE PUPIL.

By Dr. FRIEDRICH KANNGIESER, Marburg.

Abridged Translation from the *Arch. f. Augenheilkunde*, the German Edition of these Archives, lxiii., No. 1, 1909, by Dr. Matthias Lanckton Foster

MIOSIS DUE TO LIGHT AND TO CONVERGENCE.

IN order to ascertain the differences in the size of pupils contracted by convergence and by light the following means were employed. For convergence the eyes were fixed on a white knob 15*cm* away; central illumination was secured from a gaslight 30*cm* away. After waiting a minute and a half, until the pupils had become quiet, the latter were measured by means of Haab's pupillometer. A hundred persons were examined; the sex and the pigmentary condition of the iris were noted, but the refraction was not determined.

The following table shows by decades the average diameters of the pupils when thus contracted by light and by convergence as found in persons between the ages of 10 and 59 years.

Age	Light	Converg.	Difference
10–19	3.5	3.9	+0.4
20–29	3.5	3.7	+0.2
30–39	3.7	3.6	−0.1
40–49	3.3	3.1	−0.2
50–59	3.1	2.9	−0.2

These results show that from the tenth to the thirtieth year miosis from light is greater than that from convergence, while the reverse is true from the fortieth to the sixtieth year. The change comes in the thirties, at which time there is the least average difference between the miosis induced in the two ways. The decrease in the size of the pupil in response to both light and convergence with advancing age is very clear. It is also shown that sex and the color of the iris have apparently no influence on the size of the pupil.

As has been mentioned, a distance of 15*cm* was used to obtain the preceding results. Then the question arose whether the pupillary diameter could not be lessened still more by convergence on the white knob at a shorter distance. The pupils of fifty persons of either sex were measured during convergence to 15*cm*, and again during the greatest possible convergence, with the knob held as near the eyes as it could be seen distinctly. The contraction seemed to be fairly proportional to the strength of the convergence, but the following table shows that the contraction produced depended not only on the degree of convergence, but also on the age; for the difference in the size of the pupils, converged to 15*cm* and to the maximum, decreased with advancing age. Under thirty years the variation was fairly constant. Sex had no influence.

Table showing comparison between ordinary and greatest miosis due to convergence.

Age	Difference in *mm*
10–19	1.0
20–29	0.7
30–39	0.2
40–49	0.2
50–59	0.4
60–80	0.2

Form of the Pupil.

The shape of the pupil was noted in 200 persons. In only 5 cases did it vary noticeably from the normal, circular form; in four the pupils were longitudinally oval and in one transversely oval. Markedly angular pupils within physio-

logical limits were not observed, but in 12 of the 200 cases I found finely notched pupillary margins, chiefly in old people. Marked anisocoria of physiological nature I saw in only two cases, giving a frequency of 1%. Remains of the so-called pupillary membrane from the anterior layer of the iris were present in four persons, in only one in both eyes. Traces of processes from the retinal pigment layer of the iris extending into the pupil in the form of little brown membranes of fibres were present in 5% of the cases as little points on the margin of the pupil. In 2%, *i. e.*, in four of the 200 persons, they could be seen plainly.

In one case there was in each eye a fine, brown pupillary edge, formed of little segment-like processes, which caused a coarsely notched appearance. In another case there were four fine threads and a little membrane in the left pupil; in the third there were three fine brown little membranes at the edge of the right pupil and a fine network of fibres at a place on the margin of the left. The fourth and most interesting case was met with in a man 60 years old with heterochromia iridis. In the right there was only a trace of a pigment process, but in the left there were two triangular everted pigment processes which seemed to be shoved forward when the iris contracted. Between the two a connecting bridge could be seen at the pupillary margin.

COMPARATIVE STUDIES CONCERNING THE ACTION OF ATROPIN AND ESERIN UPON THE CILIARY MUSCLE AND IRIS.

The range of function of the ciliary muscle is learned from the near and far points at a given time and can be determined in young individuals without great error from the resultant refractive condition of the lens and measured in dioptries. The lens ceases to be an exact index of the range of function of the ciliary muscle with advancing age, as the increase of its curvature no longer corresponds to the relaxation of the ciliary muscle and zonule because of the commencing sclerosis of its nucleus, and the functional estimation of the contraction of the ciliary muscle from the near and far points must be faulty. An effect produced upon the ciliary muscle can therefore be determined by the near and far point method with fair accuracy only in the field of the manifest

range of accommodation present in the given individual without the administration of eserin or atropin. Theoretically the younger the individual the greater is the possibility of exact observation. An effect of atropin upon the ciliary muscle which would remove the near point in a person 20 years old from about 10*cm* to 20*cm* is incapable of demonstration in a person 40 years old because his near point is normally at 23*cm*. With this reservation the observation of the range of accommodation in the given limits parallels that of the range of function of the ciliary muscle.

The far point may be determined either by calculation from the skiascopic condition with the accommodation relaxed, or directly by artificially bringing the far point nearer with known convex lenses, or subjectively by the strongest convex lens, or weakest concave, with which the best vision can be obtained at the distance of 6*m*. The greatest possible accuracy is to be obtained from the combined use of all three methods.

The near point can be determined most easily by direct measurement, practically while reading Jaeger 1, more exactly during observation of a row of bright points which fuse into a bright line as soon as the near point has been passed. If the near point is so far away that Jaeger 1 can no longer be read it must be brought nearer artificially by means of convex glasses, and the dioptric value of these glasses must be taken into account in the calculation. It must be taken into account as a source of error that the determination of the near and the far point depends not only on the refraction of the lens, but also on the diameter of the pupil, the fluctuations of which are done away with in experiments with atropin and eserin. This could be avoided by the use of a diaphragm with an aperture equal in size to the smallest pupil during the experiments, but this would necessitate so small an aperture that it would act as a stenopaic slit and might cover up inequalities of the accommodation of the lens.

The measurements of the size of the pupils under atropin and eserin were made with the aid of Haab's scale, which is accurate to within ¼*mm*. The contraction of the pupil was fixed with paracentral acetylene illumination at a distance of 30*cm*. The dilatation of the pupil was measured

in the light reflected by a plane mirror from the weakest source of light which could illuminate the pupil. A certain time was allowed to elapse that the pupil might become quiet, or adapted, before it was measured. The experiments were performed only on emmetropes, part of them on myself, the size of whose pupils in light had been proved to be nearly constant by observations for a considerable time.

For atropin the following results were obtained: (1) The duration of action of one drop of a 1 to 1000 solution of atropin is about 100 hours, that of one drop of a 1 to 2000 solution about 60 hours; (2) the maximum dilatation of the pupil is dependent on the dose and is attained from 50 to 60 minutes after the instillation, but is maintained only a very short time. The retrogression to normal is very gradual; (3) the paralysis of the ciliary muscle reaches its maximum from 4 to 6 hours after the instillation. The point of culmination of the paralysis is therefore not congruent with the maximum dilatation of the pupil, but comes at a time when the pupil is already disposed to become smaller. Possibly this is because the ciliary muscle lies deeper than the iris and is reached later than by the atropin. Moreover the iris is acted on by the atropin mixed with the aqueous of the anterior chamber, while this mixture is diluted still more by the aqueous of the posterior chamber before it can attack the ciliary muscle and is surely somewhat weakened in its action. After the near point has been removed to the maximum, which differs greatly in individuals and is also dependent on the dose, by the action of the atropin on the ciliary muscle, it gradually returns to normal. During this rise and fall fluctuations are to be noted in which the near point may approach the normal, and again the accommodation may be so affected that Jaeger 1 can be read only with the aid of convex glasses, up to 2 D. These fluctuations are especially marked in certain individuals and are explained by the fact that every muscle with slight paresis makes convulsive efforts to produce the greatest possible contraction, due to the uneven strength of the central impulse. These are not usually manifest in the ciliary muscle, for the greatest possible tension of the latter, even in young persons, can no longer be recognized through changes in the form of the

lens, and these convulsive efforts of the ciliary muscle are to be expected in the stage of the shaking of the lens. But when an artificial paresis affects the ciliary muscle these extreme convulsive efforts may affect the form of the lens and become manifest as fluctuations of the near point. (4) The far point is scarcely affected in the atropinized eye.

For eserin the following results were obtained: (1) The effect produced by a 1 to 1000 solution of eserin lasts only about half as long as that produced by a solution of atropin of equal strength, about 50 hours; (2) the maximum effect is reached in from 30 to 50 minutes; (3) the near point is drawn nearer to perhaps half the normal. Hess explains this action as only an apparent one caused by the contraction of the pupil, but I found that by the interposition of a diaphragm with a very minute aperture before an eye without eserin the near point was drawn nearer only about 25% instead of 50% as obtained with eserin. Therefore I believe that the effect is produced by an increased curvature of the lens caused by eserin in conjunction with the artificial miosis. The fact that the maximum approach of the near point is congruent with the minimum size of the pupil favors Hess's theory, but it should be borne in mind that eserin probably is much more quickly diffused than atropin. While the effect of the latter first becomes manifest after about ten minutes, that of eserin is visible much sooner, and its maximum is attained in about half the time required by atropin; (4) as regards the far point, this was uninfluenced in many cases by a drop of a 1 to 1000 solution of eserin, except that the eye required a little more time to adjust itself from the near to the far point. Yet this immutability of the visual acuteness may have been due to the effect of the miosis, for with stronger doses of eserin the far point was distinctly drawn nearer so that concave glasses were necessary to furnish normal vision.

The combined action of atropin and eserin was also observed. A drop of a 1 to 1000 solution of eserin and a drop of a 1 to 1000 solution of atropin were simultaneously instilled. The result was that at first the quicker acting eserin caused contraction of the pupil and brought the near point nearer, but then the atropin dilated the pupil and moved

the near point away. The latter effect was not so great as when the atropin was used alone, but showed that the eserin had a certain inhibitive influence. When a drop of a 1 to 1000 solution of atropin and a drop of a 1% or 1.5% solution of eserin were given simultaneously the effect of the atropin was influenced still more and the duration of its toxic action shortened. When a drop of a 1% solution of eserin was instilled into an eye half an hour after an instillation of a 1 to 1000 solution of atropin, before the pupil had reached its maximum dilatation, it checked the dilatation of the pupil, but produced only a little effect upon the disturbance of accommodation. The effect was similar when the eserin was instilled after the pupil had attained its greatest dilatation, *i. e.*, one hour after the instillation of the atropin, and also when a drop of 1% eserin was instilled after the pupil had become dilated to the maximum by a 1 to 2000 solution of atropin. In the latter case the far point was so affected that a 3½ D. concave glass had to be placed before the eye in order to obtain the original vision, yet after about an hour full vision was present without correction. A drop of a 1 to 2000 atropin instilled at the height of the effect produced by a 1 to 1000 eserin caused a very great dilatation of the pupil, but only a moderate effect on the near point.

THE EXTENT OF THE PUPILLO-MOTOR AREA OF THE RETINA.

By Prof. F. BEST, Dresden.

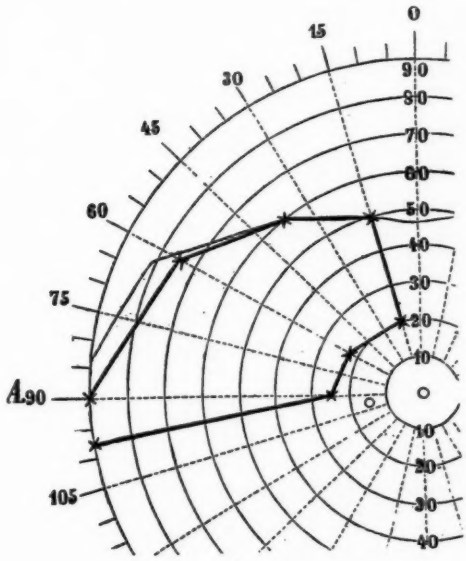
Abridged Translation from the *Arch. f. Augenheilkunde*, the German Edition of these Archives, lxi., No. 4, 1908, by Dr. Matthias Lanckton Foster.

(*With one illustration in the text.*)

THE last work of Hess has excited interest in the question whether the reaction of the pupil to light may be excited by the entire retina or by its central part alone. A pathological case in which only a small portion of the peripheral visual field was preserved, from which alone a pupillary reaction could be excited, gave me an opportunity to study the pupillo-motor power of the retina at this place.

A woman 42 years of age had a chondroma springing from the base of the skull removed from the naso-pharyngeal space through an aperture formed by chiselling away the left antrum, middle turbinate, and septum. On the next day there was a moderate œdema of the left side of the head which passed away in a couple of days. Eight days after the operation the patient suddenly remarked that she could not see with her left eye. She was positive that she could see well with it before operation. After the operation she felt a slight constant pressure upon the left eye which caused her to think it was bandaged. When she looked for the first time in a mirror she saw that the left eye was uncovered and that the absence of light perception as well as the sensation of pressure must be due to something else. Examination showed the right eye to be normal, the left amaurotic. The left eye was driven forward a trace, perhaps; not injected; movements normal. Direct pupillary reaction to light, tested only with a lamp, abolished; consensual and convergence reactions present.

Five days later movements of the hand could be recognized peripherally upward and outward. The remaining trace of visual field was quite peripheral. Illumination from above and outward caused a prompt reaction of the pupil, from all other directions it produced none. For certain reasons the examination had to be made in a room in which the window was covered by a bright curtain and the reaction was tested by reflecting the light of a lamp with an ophthalmoscope. On the next day the visual field had become larger. Although the fixation point was wanting, the field of vision was easily taken with the right eye open by cutting off the inner half of the field of the right eye by means of a screen in the mesial plane of the face. The relative position of the eyes was normal. The accompanying sketch shows the extent of the visual



field of the left eye. Colors were not recognized. The reaction of the pupil was tested in a room only slightly darkened, the same apparatus being used as in testing the field. An oil lamp to the left of and behind the patient furnished the light which was thrown by an ophthalmoscope from the arc of the perimeter into the left eye. With

the lamp burning brightly the pupil reacted from both the centre and the periphery of the retina, yet the extent of the pupillary movement was clearly greatest from the preserved field. With reduced light strength the reaction was excited by this part of the field alone, not by central illumination. During the following days the symptom complex remained the same except that the field enlarged a little so that on May 5th, twelve days after the accompanying sketch was drawn, it extended above to 15°, and outwardly to 10° from the point of fixation. Dr. Treutler now found that the central reaction was not so prompt and many times did not occur, while the reaction was present every time and most distinctly on illumination of the inner-lower portion of the retina, corresponding to the preserved portion of the visual field. On May 10th the papilla began to appear white, although in other respects everything appeared normal. The examination now was by means of a taper carried along the arc of the perimeter while the right eye was kept open for fixation and screened from the left. The brightness of the room was lessened by curtains which allowed a certain degree of light in which that of the taper excited a pupillary reaction only in the preserved part of the field, but not toward the macula. The pupillo-motor boundaries agreed with the limits of vision, except that at the periphery the pupillomotor was from 10° to 20° narrower with this light strength. The onset of the light reaction from the periphery was very dependent on the rapidity of the approach of the source of light: when the movement was much slower the reaction followed later. At the final examination on June 4th the atrophy of the left optic nerve was pronounced, otherwise everything was normal, except the pupillary reaction. The vision of the right eye was 1, that of the left was the recognition peripherally at 25*cm* of test types which should be read with normal central vision at 24*m*. Points separated 0.9*cm* could be correctly counted at 25*cm*. Color vision was defective. Blue in large masses sometimes was correctly designated, yellow and red appeared as brown, green without admixture of bright qualities seemed to be whitish. The visual field had again become smaller and was of the size shown in the sketch. Test of the field with a taper, which by diffused light gives rather larger boundaries than correspond to the seeing retina, showed an enlargement of the field outward to over 100°, inward only a little, to 15°. The pupil reaction, tested with adaptation to light, was unchanged from what has been described. In a dark room with moderate dark adaptation diascleral illumination with Wolff's mirror showed that

the light coming from all directions could be recognized if of sufficient intensity, but it was always localized upward and outward. Pupillary reaction could likewise be produced from every direction if the light was of sufficient intensity. The tumor in the nose had recurred.

To recapitulate: After extirpation of a tumor springing from the base of the skull, came an optic atrophy in which a small peripheral portion of the field was preserved while the entire centre was lost. In spite of this the reaction of the pupil to light was present and the pupillo-motor boundaries coincided with those of vision when the strength of the testing light was so proportioned to the general illumination that with the given adaptation of the eye the testing light excited a reaction from the seeing portion of the retina, but was too weak to excite a reaction when cast upon the non-seeing retina by means of diffusion from the seeing portion.

As to the pathological process that caused the optic atrophy, the following hypothesis is perhaps the most plausible. There are three possibilities: fracture of the base of the skull from chiselling out of the antrum, pressure into the left optic canal of a bit of bone already loosened by the ingrowing chondroma by the tamponade, and injury of the left optic nerve by œdema. As the chisel was not used in the removal of the tumor, and as the blindness immediately followed the operation, the second possibility is perhaps the most probable. At any rate the process was one either far back in the orbit, or, more probably, in the optic canal.

This case seems to prove that the light reflex may be excited from the periphery of the retina. Unfortunately the apparatus described by Hess was not used, because that author's exhaustive work had not appeared at the time of the examination, so objections may be raised to the exactitude of the investigation, but I believe they will not be of weight so far as the peripheral pupillary reaction comes in question. The quantity of light falling upon the retina must be exactly the same in the places to be compared. This is very nearly the case with illumination by means of a mirror carried along the arc of the perimeter, yet more accurately so in the experiment with the candle. The importance of this postulate differs greatly whether the light-adapted eye is tested in a

light room, as in our observation, or in a dark room. In the first case the test light forms only a fraction of the entire illumination which influences the pupillary movement; slight differences in the strength of the testing light cannot be of as much importance then as when the test is made in a dark room, where very slight differences in the strength of the testing light, which alone enters the eye, must cause notable errors. The same is true of diascleral light, and of the diffused light caused by the refracting surfaces in the eye. As the diffused light cannot, and the diascleral cannot readily, be excluded, it is at least to be desired that it should remain the same during the comparison of the reflex sensitiveness of two places on the retina. In the test with the candle this was nearly the case, the entire illumination of the retina remained approximately constant. It was less exact from the simple throwing in of the light by means of a mirror. It should be added that the entire illumination is increased every time by the amount of the testing light. Yet results obtained which admit of but one interpretation, provided that the test light is not too strong in proportion to the entire illumination and on the other hand strong enough to set up a reaction, show that in a test in the light the faults mentioned create no real disturbance.

Hess has further shown that the nasal and temporal portions of the retina are not physiologically equivalent, but when I obtained in this case a reaction from 30° upward and outward, but not from 30° below or from the macula, which is the strongest pupillo-motor region physiologically, influence in the production of the light reaction must be ascribed to the periphery of the retina. Deception through accommodative movements of the pupil was avoided by fixation of the right eye, sometimes by the reading of distant test types over the fixation point of the perimeter. As the condition of adaptation has an influence upon the retina it should be noticed that moderate light adaptation was always present in the test, and that no change in the total illumination took place with the exception of that caused by the test light.

Finally it is certain in this case that the pupillary reaction produced by irradiation of the peripheral seeing portion of the retina was not excited by diffused light upon the blind

centre of the retina. Although conclusions as to normal conditions must be drawn with care from pathological observations, and particularly from a single one, the generalization is justified that the periphery of the retina is pupillo-motor, for our case cannot well be explained as an abnormality. To this it may be objected that observers have not found hemianopic retinal reaction in all cases of hemianopsia, but I insist that a test in a dark room with only one very intensive light source must lead to more uncertain results than a test in a light room, in which the conditions for detecting a hemianopic reaction are favorable, as the addition to the diffused light by the test light can be neglected. Again, there have been observations in which the light reaction was found to be absent in cases of central scotoma and peripheral vision. Hess reports such a case of scotoma after retrobulbar neuritis. The pupillary reaction was almost absent and could be excited slightly only by the very strong light of an electric ophthalmoscope. It is shown that beyond doubt the periphery of the retina did not functionate normally; the vision was reduced to counting fingers when very near, while normally large letters can be recognized by the periphery not far from the macula. Color perception was also absent. The amount the nerve fibres are functionally injured by an optic neuritis varies very much, and such a case can be used in evidence only when the fibres of the periphery of the retina remain absolutely normal and when, under such circumstances, the pupil reaction is regularly absent.

But most decisive are the investigations of Hess which make it probable, according to his view, that the pupillo-motor portion of the retina can have a radius of at most 3*mm*, and that all the rest of the retina is not pupillo-motor. At the same time he says that this proposition is valid only for the light strengths used by him, and mentions in his second paper the possibility of a greater peripheral extent of the pupillo-motor area with test lights of very high light strength after he has demonstrated the gradual decrease of the excitability from the centre toward the periphery. As in our case the light strength did not exceed the limits ordinarily used clinically, which were likewise not exceeded in Hess's experiments,

a contradiction would be present if his conclusion was accepted as the only possible one. But I believe that Hess's interpretation of his experiment must not be unconditionally accepted. We must briefly review the experiment and see whether some other explanation is not possible.

1. When, with the total adaptation remaining the same, a test light is carried from a portion of the retina relatively adapted to light to one at the same distance peripherally relatively adapted to dark, no pupillo-motor activity takes place. Should the difference in the relative adaptation of the compared places in the retina be annulled, it is clear that under no circumstances could a pupillary reaction take place, because both places are equally pupillo-motor. Objectively there is no cause for the pupillary reaction except it may be the local adaptation of the retina. There are two possible explanations: Either the difference in the relative adaptation of the two neighboring places in the retina does not suffice to produce a contraction of the pupils through the preponderance of the influence of the irradiation of the test light of the relatively dark adapted place, as compared with the light adapted from which the test light is withdrawn, or these peripheral places have no pupillo-motor activity. As the case I have described shows the latter to be incorrect, there remains only the probability that differences in the relative local adaptation of neighboring places in the retina do not suffice of themselves to upset the existing pupillo-motor equilibrium. Hess expressly directs that the arc of the pupil perimeter be moved at most 10° to 20°. As perhaps greater differences of the local adaptation are possible between distant places in the retina, and as these are more independent than neighboring places, an investigation of places in the retina 180° apart, in other respects equally pupillo-motor, might be of use. Hess seems to have considered the possibility, but to have cast it aside as improbable, that the difference in the adaptation does not suffice to upset the pupillo-motor equilibrium of peripheral places in the retina. He writes that the test light seemed brighter on the dark adapted part of the retina than on the light adapted, and from the close connection between brightness and adaptation thought his theory well grounded. It should be remarked that very

intricate contrast phenomena appear. The becoming bright of the test light corresponds to a contrast darkening of its near neighborhood; and it is questionable whether the diffused light falling on the dark part of the retina could produce a local dark adaptation of importance.

2. We come now to the experiments from which Hess infers an incapacity on the part of the region of the retina about the papilla to produce a light reflex. In all of these the pupil behaved the same whether the blind spot or an equally peripheral portion of the seeing retina was irradiated. In regard to this I can present only hypotheses. We see the blind spot in our visual field in the brightness of its neighborhood, and the hypothesis is justified by the close relation between brightness and pupillary reaction that the blind spot may be filled out in a pupillo-motor sense just as in brightness. For example, if in a dark room the image of a lamplight is allowed to fall on the optic nerve no lamplight is perceived, but a diffused brightness, and if now a place above or below equally peripheral is fixed I can hardly note an increase of the total brightness; this is true also if the intensity of the light is very low. The presumption is a quick change of fixation to avoid weariness. If the light is still further decreased it can be perceived only when it falls on the seeing retina, but it is then too weak to produce a pupillary reaction. From all of which I assume that there is a compensation as regards the optic papilla in its pupillo-motor ability as in the visual field. How, I do not know. There are too many possibilities that cannot be excluded with our present knowledge.

To sum up. Hess has shown that the pupillo-motor function of the retina gradually decreases from the fovea outward in the eye adapted to light, corresponding to the peripheral diminution of vision and of color perception. My observation indicates that the periphery of the retina is reflex sensitive to intensities of light clinically used. The analysis of Hess's experiments shows that they do not disprove this peripheral reflex sensitiveness.

I also believe that the determination of the isokinetic lines upon the retina in the way made by Hess to an excentricity of 30° approximates the real pupillo-motor value of the

periphery of the retina, provided that the test lights used form only a very small fraction of the total illumination. In this case the diffused light from the test light is relatively immaterial. The determination would be still more exact if the isokinetic lines were measured with different degrees of high and low illumination, whence conclusions might perhaps be drawn concerning the true pupillo-motor value of peripheral parts of the retina. Further, the determination of the isokinetic lines farther toward the periphery might show that the motor excitability of the far periphery temporally is quicker than that nasally, which could be explained only by the theory of an excitability of the periphery itself.

Finally I would add a few words in regard to the existence of special pupil fibres. The dependence of the pupil reaction upon adaptation and brightness speaks strongly for a close anatomic connection. In diseases of the optic nerve and retina the parallelism between brightness and pupillary reaction is always present. In retrobulbar neuritis I have never seen blindness associated with a preserved pupil reaction, and in the gradual return of vision there has always been a corresponding pupil reaction, though often difficult to demonstrate. As one never knows how many fibres no longer functionate in the optic nerve, and as probably the intensity of the reaction is dependent on the entire number, there can be places with moderate vision and bad reaction; and, on the other hand, through evenly distributed disturbance of fibres, bad vision with moderate pupil reaction.

I may briefly report here a case of retrobulbar neuritis in which the pupil reaction ran parallel to the vision throughout, as it is of interest also as regards prognosis. A man 45 years old came under treatment, June 28, 1907, on account of acute retrobulbar neuritis of the right eye. He had a large central scotoma with normal peripheral boundaries of the field and reduced direct pupillary reaction. The vision gradually decreased until on July 6th there was total amaurosis which persisted for nine days, with no direct light reaction. The left eye became affected and gradually grew worse until on July 18th there was total blindness for one day. Then the vision gradually began to return in both eyes. In September it was for the most part restored, but

continued to improve slightly until February, 1908, since which time it has remained stationary at $\frac{5}{7.5}$, somewhat better in the left than in the right. Objectively there was for a short time a hyperæmia of the optic nerves. The right eye was myopic 10 D., the left 6 D. The right eye had 0.5 D. of regular astigmatism, the left 1.25 D. The right had a large circular staphyloma, the left a small one. The cause of the retrobulbar neuritis was rheumatism.

To return to our subject. There are observations published in literature in which the pupillary reaction was preserved though vision was lost through disease of the optic nerve. It is questionable whether an error is not present in these very rare exceptions, as the blind often erroneously claim a trace of light perception and inversely patients declare themselves blind who have a certain degree of light perception or of vision. But even if these observations are correct they do not prove the existence of special pupil fibres. It is not certain that the fibres of the optic nerve have but one kind of function. The optic nerve fibres may have different kinds just as the peripheral receptive have in spite of their minuteness. It cannot now be decided from physiological or pathological observations whether there are special pupil fibres or not.

A NEW METHOD OF EXAMINATION FOR DOUBLE IMAGES.

By Dr. WALTER HESS, Zürich.

Abridged Translation from the *Arch. f. Augenheilkunde* (the German Edition of these Archives), vol. lxii., Nos. 2–3, 1908, by Dr. Matthias Lancketon Foster.

(*With two illustrations in the text.*)

A CERTAIN inexactitude in the methods hitherto used in the investigation of double images, the relatively great dependence on the intelligence and good will of the patients, and the difficulty of preserving exact notes have induced me to seek a method free from such objections. The following is the result of my endeavor.

Four broad green lines are drawn on a black chart so as to form a quadrilateral. The patient is provided with a red glass over one eye, a green one over the other. The examiner holds a black rod with a red mark at its end. The green lines and the red mark will both be seen by the patient, but not with the same eye, because the effect of each colored glass is to efface the complementary color into the black background. In the examination the patient is directed to follow the red mark as the physician moves it over the chart and to state when it crosses one of the green lines. This will be stated correctly when the two eyes have normal projection. A patient with diplopia will perceive the red mark as displaced from the line when it is in reality upon it, and will perceive it as on the line when in reality it is not, evidently at such an angle of deviation as corresponds to the angle of deviation of the two visual axes, for the eye following the red mark receives its impression in the macula so that the

image of the green line must fall on the macula of the other eye when the red mark seems to fall on the line. If, further,

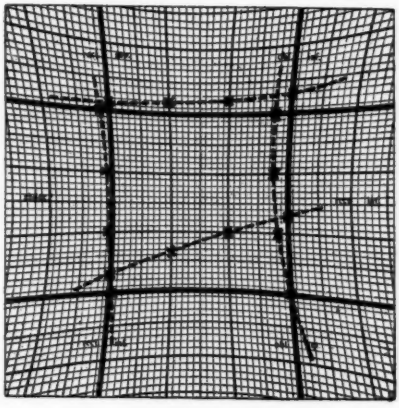


Fig. 1a.

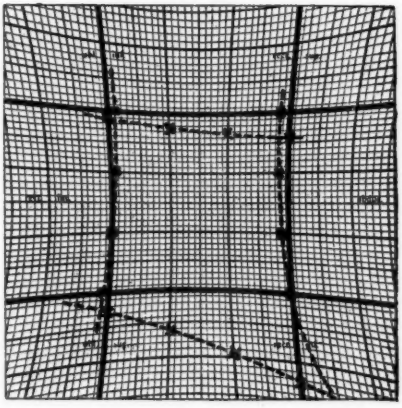


Fig. 1b.

the places where the red mark seems to cross each line are determined, the relative positions of the eyes when looking in

different directions are learned, and thereby the functional activity of the muscles.

The following is a concrete example. (See Figs. 1a and 1b. The diagnosis was paralysis of the left superior oblique. In Fig. 1a the broken lines indicate the course of the red mark and the patient's projection of the solid green lines with the red glass before the left eye. Fig. 1b similarly shows the projection of the green lines when the red glass was before the right eye.) The points where the red mark seemed to cross the green lines are marked with x. The signification of the lines thus formed is that they form a design of all the fixation points of the eye covered with the red glass when the eye covered with the green glass looks toward points in the corresponding green lines; also that they surround a surface which includes all fixation points of that eye when directed to points in the field bordered by the green lines. The reciprocal relations of the red and the green fields thus correspond in size, form, and direction exactly to the simultaneous relations of the fields of the two eyes covered with red and with green. The marks obtained form a direct, graphic delineation of the positions and functional movements of the two eyes within a certain area.

Two different delineations may be obtained by placing the red glass first over the right eye and then over the left. If the red glass is placed before the affected eye the delineation is called primary (Fig. 1a) as the regular green figure corresponds to the sound eye. The red figure is smaller than the green, corresponding to the restriction of movement of the affected eye. When the glasses are changed we obtain the secondary delineation (Fig. 1b); the green field now appertains to the affected eye while the red field is larger, corresponding to the greater functionating capacity of the sound eye. We can use both delineations for the exact diagnosis of the muscle or muscles affected. Both should be taken in every case as the examination of them will show which is the paralyzed eye, as it will have the smaller field in each, and the comparison of the two always forms a control by which errors and attempts at simulation may be exposed. The two must contrast in every way. When in one, *e. g.*, the red field is contracted from the right and downward and is drawn to the

left, in the other it must be enlarged to the right and downward and drawn to the right.

In later examinations, to determine gradual improvement, it is necessary to take only the primary, the one in which the red glass is placed before the paralyzed eye.

The black chart, green lines, and red mark are made of felt and wool in order to avoid all reflection and obtain the most complete possible disappearance of the complementary colors. The dimensions of the chart are 120*cm* x 120*cm*. It does not seem advisable to make it smaller than this because when there was a great deviation of one eye the projection of the mark would fall outside the chart and could be perceived with difficulty. Besides the results are more accurate when the field bordered by the green lines is rather large. A larger chart would be valuable in some cases for these reasons, but is rather inconvenient.

The ruling on the chart corresponds to the projection of angles upon a plane at the distance of 1*m*. In order to maintain this distance conveniently a thread of this length is extended from the centre of the chart to the root of the nose of the patient. The green bands are then 30° from the centre, the ruled lines, which serve for the placing of the red mark, 10° and 1° respectively. In the examination it is best to determine four points of crossing for each line, as seen by the patient on the chart, and connect them. More than four points are at most of only theoretic value, while two points may suffice, especially in the later examinations, to ascertain whether there has been improvement or the reverse. During the examination, movement of the patient's head must be avoided. This is easily accomplished by having him lean his head forward into a rest, similar to that used by photographers. Absolute fixation of the head is unnecessary, as when the head is leaned forward in the manner described the slightest movement is easily perceived both by the physician and the patient and controlled.

Before the red-green glasses are placed on the patient he should be instructed to indicate the instant the mark crosses the line and that he may correctly understand what is meant it is well to have a preliminary test with one eye closed without glasses, when naturally the observation of the patient will be

in accordance with the truth. If with the glasses on the patient fails to perceive the red mark, his attention is called to it by moving it here and there over the black chart, and then he is directed to follow it wherever it is moved along the system of ordinates. In some cases it is impossible to obtain crossing of the lines in certain parts of the chart, *e. g.*, when one eye is so limited in its movement that it cannot follow either the lines or the mark to the periphery. In such cases one has to be satisfied with the determination of points of crossing which lie in the area common to the two eyes. The figure constructed from points so obtained will be incomplete, as it portrays the direct response of the function of movement, which may be limited to any degree up to total paralysis. The differentiation which muscle or muscles are involved in the paralysis is given when one knows their functions; for the primary delineation, a contraction in the direction or directions in which there is a deficient movement, and at the same time there is a displacement of the field in toto in the direction in which the diseased eye is deviated by the preponderating action of the antagonists. This suffices for the diagnosis of all muscular paralyses when the active components that produce movement in the horizontal and vertical meridians are known. The action of the superior and inferior recti is most easily differentiated from that of the superior and inferior obliqui by the fact that the former raise and lower the eye in the temporal portion of the field while the latter lower and raise it in the nasal, so that the red field will be contracted in the one case upward or downward and outward, in the other downward or upward and inward. Paralysis of the abducens or of the internal rectus causes a contraction outward or inward. To make the diagnosis easy the name of each muscle may be printed on the chart in the area governed by its function, so that the diagnosis may be directly read off. Then a glance will suffice to recognize quantitatively and qualitatively the paralyses present even when they are combined.

This method may likewise be used in concomitant strabismus, provided of course that neither eye is too amblyopic. Results both theoretically and practically important may be obtained if we make such a perfectly exact picture of the

position and functional activity of the eyes before and after an operation for strabismus. The objective demonstration of the false projection induced by prisms, and of the convergence accompanying accommodation when concave glasses are placed before the eyes, are instructive. The advantages of the method may be thus summed up:

It furnishes simply and quickly an exact, direct, graphic presentation of the positions and movement functions of both eyes. Errors through tendency to fusion, faulty comprehension of the patient, or his ill will, are as good as excluded and can always be recognized. The light impression which the patient has to regard falls in both eyes on the fovea, so that his statements are not only more precise than when he has to consider a peripheral image, but they may be obtained even when we have to deal with a very great deviation or an already suppressed image.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD AND FOURTH QUARTERS OF THE YEAR 1909.

By Dr. G. ABELSDORFF, in Berlin; Prof. ST. BERNHEIMER, in Innsbruck; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin; with the assistance of Prof. A. ALLING, New Haven; Prof. E. BERGER, Paris; Prof. CIRINCIONE, Genoa; Dr. DALEN, Stockholm; Prof. HIRSCHMANN, Charcow; Dr. J. JITTA, Amsterdam; Mr. C. DEVEREUX MARSHALL, London; Dr. H. MEYER, Brandenburg; Dr. P. VON MITTELSTÄDT, Metz; Dr. H. SCHULZ, Berlin; Prof. DA GAMA PINTO, Lisbon; and others.

Translated by Dr. MATTHIAS LANCKTON FOSTER.

(*Continued from page 140.*)

Sections XIII.–XVIII.—Reviewed by Dr. NICOLAI, Berlin.

XIII.—LENS.

312. ZANI. **Clinical contribution to the etiology and pathogenesis of congenital luxation of the crystalline lens.** *Annali di ottalmologia*, 1909, xxxviii., fasc. 4.

313. KIPP, C. J. **A case of congenital coraliform cataract of both eyes.** *Amer. Jour. of Ophthalmology*, August, 1909.

314. HANDMANN, M. **Concerning the beginning of senile cataract in the lower portion of the lens.** *Klin. Monatsbl. f. Augenh.*, xlvii., ii., 6, p. 692.

315. MAWAS, J. **Contribution to the study of the pathogenesis of cataract in general and of senile cataract in particular. The lesions of the ciliary body in senile cataract.** *Rev. gén. d'ophtalm.*, xxviii., p. 481.

316. WÜRDEMANN, H. V. **The expression of cataract in its capsule.** *Jour. Amer. Med. Assoc.*, Sept. 4, 1909.

317. GREENE, D. W. **Experiences in the expression of cataract in the capsule by the Smith method.** *Jour. Amer. Med. Assoc.*, Sept. 4, 1909.

318. CLUCHIE, N. G. **A method of cataract extraction with the retention of a corneal-conjunctival bridge.** *The Lancet*, 9, x., 1909.

319. HOËG, N. **Contusion cataract.** *Hospitalstid*, 1909, p. 347.

320. HESS. **Cataract and secondary cataract operations.** *Sixteenth International Congress at Budapest*, Aug.–Sept., 1909.

321. LAGRANGE, FELIX. **Subconjunctival luxation of the lens upward and inward; migration into the cul-de-sac downward and outward.** *Arch. d'ophtalmologie*, xxix., p. 753.

322. JURAI. **Casuistics of circular opacity of the anterior surface of the lens (Vossius).** *Zeitschrift f. Augenheilkunde*, xxii., p. 126.

According to ZANI (312, **Clinical contribution to the etiology and pathogenesis of congenital luxation of the crystalline lens**), spontaneous congenital luxation of the lens is fairly rare, not more than 135 or 140 cases having been observed as yet. The patients observed are always intelligent and well developed children; cases with other developmental faults are exceptional. Various ingenious theories have been advanced to explain its origin. Zani reviews these theories and inclines to that of Badal as completed by Lagrange. According to this all eyes with such an anomaly are myopic, and on account of the form of the eyeball the lens is small in size as compared with the space it should occupy. The overstretched zonula yields and the lens is displaced toward the side in which there is the least resistance. He speaks finally of the great miosis in his case, which he ascribes to an attempt on the part of Nature to reduce to a minimum her own fault by an ideal stenopaic slit. CALDERARO.

In KIPP'S (313, **A case of congenital coraliform cataract of both eyes**) case immediately behind the anterior capsule there extended through the entire thickness of the lens an opaque core not over 1*mm* in diameter from which there radiated in all directions through its course opaque spokes ending in trumpet-shaped enlargements. Between the spokes were small glistening silvery points. The formation had the exact appearance of coral. There was no hereditary influence. ALLING.

HANDMANN (314, **Concerning the beginning of senile cataract in the lower portion of the lens**) has made notes concerning the lenticular opacities in 845 cataractous eyes and finds that the cataract usually begins below and to the nasal side, and progresses from the cortex backward and to the equator more frequently than from the cortex forward. The cause of this can be only disturbances of nutrition which

become evident sooner in the lower segment. Thus pathological products will sink and cause more injury in the deeper strata than in the upper part of the eye, and the upper lid favors this as a protective covering for the upper portion of the lens. In diabetic and glaucomatous cataract the conditions appear to be different.

MAWAS (315, **Contribution to the study of the pathogenesis of cataract in general and of senile cataract in particular**) reports the anatomical examination of the eye of a man 68 years old which was enucleated because of commencing sarcoma of the choroid and had an incipient cataract. He found a sclerosis of the ciliary body and formation of vacuoles in the cells of the pars ciliaris retinæ. He thinks that all cases of cataract are due to changes in the ciliary body that result in a disturbance of the secretion of the aqueous, and that the pathological changes in the lens capsule and in the epithelial cells of the posterior surface of the anterior capsule are caused by the chemically altered aqueous. BERGER.

WÜRDEMANN (316, **The expression of cataract in its capsule**) has operated upon 40 cases by this method. There were 10 in which vitreous was lost and four absolute failures from different causes. There is a prevalent feeling that the Hindoos upon whom Major Smith operates are more favorable subjects than ours. ALLING.

GREENE (317, **Experiences in the expression of cataract in the capsule by the Smith method**) gives statistics of 75 extractions. His experience is favorable to the operation. There were 13 cases of loss of vitreous with two failures therefrom. The capsule was ruptured four times with retention of the capsule in two of them. The average vision for 72 was better than $\frac{20}{30}$. ALLING.

CLUCHIE (318, **A method of cataract extraction with the retention of a corneal-conjunctival bridge**). Instead of completing the section of his conjunctival flap, Cluchie cuts a narrow band 4*mm* broad by 12*mm* long and leaves it in situ.

Unfortunately this operation was suggested and performed by Desmarres in 1851 (Desmarres, *Traité des maladies des yeux*, 1858, iii., 2d edition, S. 255 *et seq.*, quoted in Czermak's *Augenärztliche Operationen*, 2d edition, vol. ii.,

page 548.) He even suggests, as Cluchie does, that if difficulties ensue the bridge can be divided with scissors.

T. HARRISON BUTLER.

HOËG (319, **Contusion cataract**) reports four cases of changes in the lens following contusion of the eyeball. In two cases the anterior capsule was ruptured, in one there was a very transient cloudiness of the anterior capsule, and in the fourth case there was a ring-shaped opacity of the anterior surface of the lens. He does not believe the general assumption that this opacity is caused by the immediate pressure of the cornea upon the lens, but that the shock is transmitted through the aqueous from the cornea to the iris and the anterior surface of the lens. HELLGREN.

HESS (320, **Cataract and secondary cataract operations**) considers that the operation for cataract without iridectomy gives the best result because the pupil is preserved. But he fears the danger of a prolapse of iris and therefore excises a small piece near the root of the iris. He has tested this method in about 1000 cases and prefers it to all other methods. Concerning the time when the operation should be performed, he considers the question whether the cataract is ripe or unripe of less moment than whether the loss of vision incapacitates the patient for work. Removal of the lens in high myopia frequently leads to complications and should therefore be performed only in special cases in *one* eye. For secondary cataract an incision in the sclera and removal of the mass of after-cataract are recommended.

LAGRANGE (321, **Subconjunctival luxation of the lens upward and inward; migration into the cul-de-sac downward and outward**) observed a traumatic subconjunctival luxation of the lens upward and inward in a woman 35 years old; five days after the traumatism the lens was beneath the conjunctiva downward and outward. He thinks the migration of the lens was induced by the contraction of the ocular muscles. BERGER.

JURAI (322, **Casuistics of circular opacity of the anterior surface of the lens**) has collated the reported cases of ring-shaped opacity on the anterior capsule following contusion, and adds three more cases, making in all 17. The complications were hyphæma, subconjunctival hemorrhage, con-

junctivitis, mydriasis, rupture of the sphincter, iridodonesis, posterior synechiæ, cloudiness of the retina, hemorrhage at the macula, rupture of the choroid, and extravasations in the iris. In the uncomplicated cases the vision remained normal, the opacity disappeared in from 1 to 6 weeks. In one case complete opacity of the lens appeared later and led to extraction.

XIV.—IRIS.

323. JENNINGS, O. W. **Etiology of iritis.** *Ophthalmology*, Oct., 1909.

324. BERARD. **A case of iridocyclitis of infectious origin cured by Roux's serum.** *Revue d'hygiène et de thérap. oculaires*, April, 1909.

325. GILBERT. **Etiology and histopathology of traumatic uveitis.** *Meeting of the Soc. of Morphology and Physiol.*, Munich, Nov. 16, 1909.

326. HEERFORDT, C. F. **"Febris uveo-parotidea subchronica" localized in the parotid gland and the uvea of the eye and frequently complicated by paresis of the cerebro-spinal nerves.** *Arch. f. Ophth.*, lxx., 2, p. 254.

327. GOLDBERG, H. G. **Flat sarcoma of the uveal tract.** *Annals of Ophthalmology*, Oct., 1909.

328. RADCLIFFE, M. **The choice of operation for iridotomy.** *Annals of Ophthalmology*, Oct., 1909.

329. DUFOUR, M. **On cases of difficult iridectomy.** *Ophthalmoscope*, August, 1909.

JENNINGS (323, **Etiology of iritis**) has studied the hospital records of five hundred cases of non-traumatic iritis and finds the percentage of cases as follows: syphilis, 61%; rheumatism, 25%; gonorrhœa, 5%; influenza, 1%; tuberculosis, 1%; malaria, 1%. Numerous other causes are assigned, but each has less than 1%. ALLING.

BERARD (324, **A case of iridocyclitis of infectious origin cured by Roux's serum**) obtained improvement after few injections; after the third the vision improved and the pain became less. From this experience he would recommend early serum treatment, but further experience is necessary as conclusions cannot be drawn from one case.

GILBERT (325, **Etiology and histopathology of traumatic uveitis**) examined bacteriologically and pathologically 21 eyes with deep infectious inflammation following traumatism. The bacteriological examination gave a negative result in 12, a positive in 9. The most prominent among the pyo-

genic germs was the pneumococcus; the saprophytes are of less importance etiologically. In the histological examination comparisons were made between the special form of traumatic uveitis and sympathetic inflammation which show that the pictures are often exactly alike and that in debatable cases the histological examination cannot be conclusive. Sympathetic ophthalmia is pre-eminently a clinical diagnosis.

HEERFORDT (326, **"Febris uveo-parotidea subchronica" localized in the parotid gland and the uvea of the eye and frequently complicated by paresis of the cerebro-spinal nerves**) reports 3 cases of parotitis which ran a course not quite normal, associated with an iridocyclitis; pareses were present in 2 cases and optic neuritis was also observed. It is questionable whether this should be called an epidemic parotitis with atypical course; it is perhaps justifiable to look upon this symptom complex as unique and to look for the etiological factor. Probably the microbes are similar to those met with in epidemic parotitis.

GOLDBERG (327, **Flat sarcoma of the uveal tract**) found the entire uveal tract infiltrated with a spindle-celled sarcoma in an eye which was enucleated for secondary glaucoma. In another case an angio-sarcoma was found in the socket of a patient whose eye had been enucleated five years before. Both eyes had suffered from traumatism. ALLING.

RADCLIFFE (328, **The choice of operation for iridotomy**) recommends the Ziegler V-shaped incision and relates seven cases which illustrate the good results he has obtained.

ALLING.

DUFOUR (329, **On cases of difficult iridectomy**) has in difficult cases operated by Gayet's method. He opens the anterior chamber by cutting from without inwards, and then enlarges the incision each way with two small, bent, round-pointed knives. The first incision is performed with a scarificator. When the anterior chamber is so shallow that transfixion is impossible Gayet's procedure is very valuable.

T. HARRISON BUTLER.

IN MEMORIAM.

Dr. Charles J. Kipp.

The New York Ophthalmological Society has heard with profound regret of the death of Dr. Charles J. Kipp, one of its oldest members.

Dr. Kipp was born in Hanover, Germany, on Oct. 22, 1838, and died of pneumonia after a brief illness on Jan. 13, 1911. He came to the United States at the age of sixteen. He was graduated from the College of Physicians and Surgeons, New York City, in the class of 1861. Shortly after his graduation he joined the Army as Assistant Surgeon and served throughout the Civil War. In 1864 he was promoted to be Surgeon with a brevet rank of Lieutenant-Colonel. He resigned from the Army in 1868 and devoted himself to the study of eye and ear diseases. Settling in Newark, he opened the outdoor department for eye and ear diseases in St. Michael's Hospital. Later he founded the Newark Eye and Ear Infirmary, where he filled the position of Executive Surgeon until his death. Though living at a distance he regularly attended the meetings of this Society and took an active part in its proceedings. From 1885–1886 he was its President.

His sterling qualities, his painstaking care, his sincerity and kindliness endeared him not only to his friends, but to the public at large. The Profession bestowed on him its highest honors. He was at one time President of the Medical Society of the State of New Jersey; one of the Vice-Presidents

of the American Medical Association; from 1907–1908 President of the American Ophthalmological Society; later President of the American Otological Society.

His contributions to medical literature were numerous and valuable. He was the first to recognize that otitic thrombosis of the lateral sinus was frequently accompanied by optic neuritis. In 1876 he read a paper before the International Ophthalmological Congress held in New York on Subconjunctival Cysticercus, probably the first case ever described in America.

This Society loses in the death of Dr. Kipp a staunch and energetic friend, an experienced and indefatigable worker; one whose qualities of heart and mind won the affection and respect of all who came in contact with him.

In commemoration of his useful and beautiful life, your committee recommend that this "In Memoriam" be spread upon the minutes of the Society, and that a copy be sent to his next of kin and to the Medical Press.

EMIL GRUENING.
THOS. R. POOLEY.
PETER A. CALLAN.

NOTICE.

SCHOLARSHIP IN OPHTHALMOLOGY.

A Scholarship in Ophthalmology has been founded by William A. DuBois, Matthew B. DuBois, and Katherine DuBois, sons and daughter of Dr. Abram DuBois, who have presented to Columbia University the sum of $18,000 to be used as a fund for this purpose. This is to be known as "The Doctor Abram DuBois Memorial Fund." The scholarship is open to graduates of the College of Physicians and Surgeons, who have satisfactorily completed a term of service in an Eye Hospital and who have given evidence of serious intention of pursuing ophthalmology as a specialty. The holder of the scholarship is expected to devote himself to post-graduate studies, preferably of a scientific character,

connected with ophthalmology in foreign and American universities. The income amounts to about seven hundred dollars ($700) a year.

The bestowal of this scholarship is in the hands of a committee consisting of the Professor of Ophthalmology, the Dean of the Medical School, and Dr. Edward L. Partridge.

IV.—**Lehrbuch der Augenheilkunde (Text-book of Ophthalmology).** By Prof. T. AXENFELD, Freiburg, Germany, in collaboration with Professors Bach, Bielschowsky, Elschnig, Greeff, Heine, Hertel, v. Hippel, Krückmann, Oeller, Peters, and Stock. Second Edition: G. Fischer. January, 1910. Price, Mk. 14.

This text-book, which was reviewed favorably in these ARCHIVES last year, now appears in a second edition. It has gained principally by the addition of Oeller's beautiful fundus pictures. These comprise eleven colored plates. The text of the book has been revised so that greater uniformity has been obtained. The striking feature of the book remains the great number and excellence of the text illustrations; notwithstanding, the price is unchanged and very reasonable.

A. K.

V.—**The Prescribing of Spectacles.** By A. S. PERCIVAL, Newcastle-on-Tyne. Pages, 159. Publishers: John Wright and Sons, Ltd., Bristol, England. 1910. Price, 5/6 net.

This is an extremely practical introduction and guide to the study of errors of refraction and motor anomalies. The optical section, 50 pages, gives the necessary mathematics required in optical problems. The combination of the theoretical and practical in the same book, though distinctly separating the two, is excellent, and should meet the favor of all classes of readers.

A. K.

VI.—**Ophthalmic Therapeutics.** By Dr. A. DARIER, Paris. Translated by Sydney Stephenson, London. One of the volumes comprises "An International System of Ophthalmic

Practice." Edited by Dr. W. C. Pyle. Pages, 443. Publishers: P. Blakiston's Sons and Company, Philadelphia. 1910. Price, $4.00.

The subject of Ocular Therapeutics is one in which the author has been particularly interested for many years and on which his writings, "Leçons de Therapeutique Oculaire," have long been favorably known. In the present treatment of the subject, the work is divided into two parts: General Therapeutics and Special Therapeutics. In the first part, on General Therapeutics, the following subjects are fully described: Methods of Diagnosis, Constitutional Treatment, Extraoral Treatment, Intraocular and Subconjunctival Injections, Serum Therapy, Diaphoretics, Phototherapy, Electricity, X-Ray, Radium, Hydrotherapy, Local Medication, Anæsthetics, Analgesics, Vasodilators, Vasoconstrictors, Mydriatics, Miotics, the Silver Compounds. In the second part, Special Therapeutics, each disease of every anatomic subdivision of the eyeball and its adnexa is taken up in turn and its treatment is given.

The work of the translator is excellently done, though Dr. Darier's characteristically subjective style cannot but lose in a foreign language. The reader will find the standard methods of treatment well described and many suggestive ideas.

A. K.

VII.—**Lessons on the Eye, for the Use of Undergraduate Students.** By FRANK L. HENDERSON, St. Louis. Fourth edition, revised. Pages, 238. Philadelphia. Publishers: P. Blakiston's Sons and Company. 1910. Price, $2.00.

An excellent little book, one that admirably serves its purpose. There are, however, omissions of subjects which to the reviewer's mind are important for the student to know, such as visual acuteness and the field of vision and their methods of examination. The space occupied by cuts of certain instruments, such as on page 190, eye speculum, etc., could be spared. Many of the illustrations are good, though the fundus pictures do not convey much meaning, nor are those of interstitial keratitis, page 133, or congestion of iris, page 154, instructive.

A. K.

VIII.—**The Ophthalmic Year Book.** Volume VII. By E. JACKSON, T. B. SCHNEIDEMAN, and W. ZENTMAYER. Illustrated. Pages, 393. The Herrick Book and Stationery Company, Denver, Colo. 1910. Price, $5.00.

Volume VII. of this Year Book brings reviews of important articles on ophthalmology and a complete bibliography for 1909.

As one of the editors, Dr. W. Zentmayer replaces Dr. de Schweinitz. The high standard of previous years has been fully maintained and the book remains invaluable to every one interested in the science of ophthalmology.

A. K.

IX.—**Glaucoma.** An Inquiry into the Physiology and Pathology of the Intraocular Pressure. By THOMAS HENDERSON, M.D., Nottingham. Longmans, Green & Company, 30th Street and Fourth Avenue, New York. 1910.

The book is dedicated to Leonard Hill, with whose aid many of the experiments were made. Reasoning from the analogy between the intraocular pressure and intracranial pressure the author believes that the former depends on the intravenous pressure. After showing the differences in the angle of the anterior chamber in eyes hardened in Müller and in formalin, the importance of a truly radial microscopic section is noted to give a correct topographical picture of this region. The cribriform ligament, a term which the author prefers to pectinate ligament, is shown to arise from the deepest corneal lamellæ and not from Descemet's membrane. This ligament undergoes changes in life, and in old age becomes fibrous, a sclerosis which may be of pathological significance. The ciliary processes, the iris crypts, and the return venous flow with Schlemm's canal are carefully described, based on the author's original and very interesting investigations. As to intraocular pressure, the author believes that the corneo-scleral envelope is rigid, and that the intraocular volume is a fixed quantity. The intraocular pressure is vascular in origin and is influenced by the venous pressure. There is thus a parallelism between the intraocular and intracranial contents. One of the functions of the iris according to Henderson is to absorb the aqueous through

its crypts and veins. The venous nature of the circulus iridis major, aside from its anatomical foundation, receives physiological support from the fact that this additional arterial supply to the iris is unnecessary for its function. The author then considers the pathology and treatment of glaucoma. The value of an iridectomy is seen in the raw edges of the coloboma permitting access of the aqueous to the iris veins. The importance of general medical treatment is emphasized. Though many of the author's deductions in the last half of this book require further proof, Dr. Henderson has unquestionably furnished us with the most valuable contribution to the subject of glaucoma in many years.

A. K.